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DISEASES

of the

CHEST

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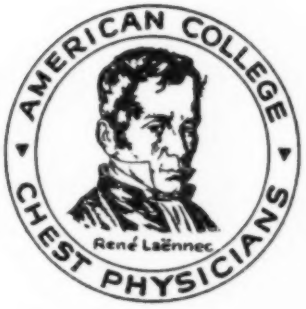

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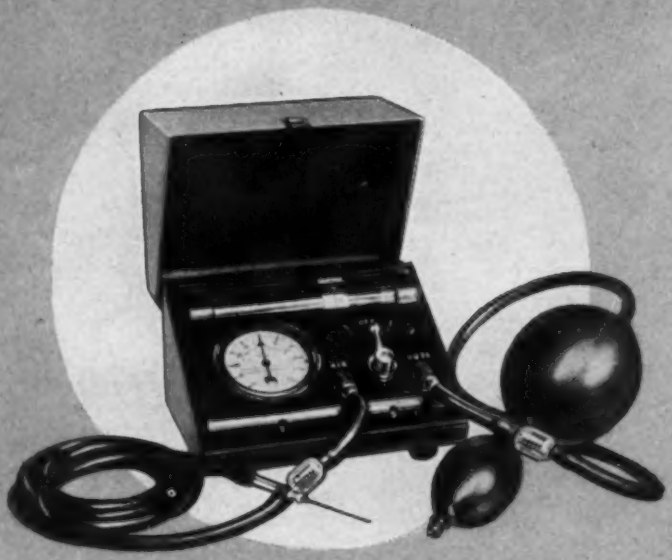

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One complete excursion of the piston delivers 150 cc of air. Turning the valve immediately starts the piston in the other direction, continuing to supply air without any change of connections. Air can be delivered either slowly or rapidly as desired. An aneroid manometer is in the circuit at all times, indicating air pressure during instillation as well as the pressure in the chest when the valve is turned to zero.

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DISEASES *of the* CHEST

VOLUME XIV JULY-AUGUST, 1948 NUMBER 4

The Role of Trauma in Initial Pneumothorax

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INTRODUCTION

That injury to the lung can occur while the initial pneumothorax is being induced has been recognized since the introduction of this therapeutic procedure. Forlanini,[1,2] Lillingston,[3] Murphy,[4] and Lemke[5] all commented on the possibility of lung puncture during induction while Brauer,[6] and Parry Morgan,[7] were convinced that it was difficult to avoid injury to the lung during the operation.

Nevertheless, the early workers in this field were not unanimous in the recognition of the frequency of lung puncture during initial pneumothorax and the majority believed, with Robinson and Floyd,[8] that a careful operator could avoid injuring the lung. It has been the optimism of the latter that we have inherited. This is embodied in the widespread present-day acceptance of the puncture technique for initial pneumothorax. At the present time, it is generally accepted that induction of pneumothorax consists of inserting a needle through the chest wall, penetrating the parietal pleura and then, with the two layers of the pleura separated by the needle tip, introducing air into the space between. This is not considered difficult.

This confidence in the simplicity of the operation is not completely shared by a number of clinicians. Fishberg, for example, noted that, "The main difficulty is to pass the needle as far as the costal pleura, puncture it and avoid penetrating the visceral pleura and the lung."[9] Barnwell, too, complains that, "The hand is not sensitive enough to tell whether the one or both layers of the pleura have been traversed except when the two layers are separated by air or fluid."[10] W. Parry Morgan declared in 1914,

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that *every* initial pneumothorax involved trauma to the lung and, indeed, that it was only because of this injury that it was possible to establish a pneumothorax space at all.[7] Unfortunately, this hypothesis was not supported by experimental data and, subsequently, other explanations were offered for the "accidents" not infrequently observed during induction of pneumothorax.

Knopf[11] regarded excessive collapse at induction as due to the expansion of nitrogen in the warmer confines of the thorax while Balboni[12] regarded it as due to the rupture of an adhesion. Beggs[13] considered that the loss of support of the chest wall caused weak points in the visceral pleura to give way while Mattill and Jennings[14] suggest that this might happen at a caseous area. Bronfin[15] and Pollack[16] ascribe the accident to the rupture of a bleb. None of the above workers, with the exceptions of Mattill and Jennings, considered pulmonary trauma as an important factor in the etiology of "spontaneous" pneumothorax following initial pneumothorax.

The question remained unsettled until 1936 when one of us (I.G.T.), by clinical experiments, independently reached conclusions that seemed to confirm the hypothesis of Parry Morgan.[17] In each of 17 consecutive patients, the routine method of the pneumothorax induction was employed except for one important modification: *after pleural readings were obtained the needle was withdrawn and no air was given. In each case subsequent fluoroscopy and roentgenological study showed a pneumothorax space to be present.* It was noted, however, that this space was of gradually increasing size for a period of several hours, reaching equilibrium after approximately twelve to twenty-four hours. Analysis of the gases contained in several of these "spontaneously" appearing pneumothorax spaces showed them to be similar to alveolar air in composition. It was concluded, "A small puncture (of the lung) is produced and a variable amount of air is liberated in the pleural space producing a small air pocket. This is the real initial pneumothorax. It is into this pocketed air that we introduce the larger amounts producing by successive refills collapse of the lung." This concept received experimental support recently when Ornstein, Herman and Friedman[18] measured the volume present in pleural cavities 24 hours after the induction of artificial pneumothorax. In each of the five cases studied, "... There was an increase in the volume of gas over the volume of air injected at the primary induction of the pneumothorax."

The above quoted studies of Tchertkoff were performed with sharp-pointed pneumothorax needles. Although controversy over the type of needle to be used has abated considerably during the past two decades,[19] it was recognized that the needle used may have influenced the results. Indeed, it has been claimed that

whereas pulmonary trauma might occur with sharp needles, the use of dull points provides a safeguard against lung puncture. Edgar Mayer states that, "With a blunt needle these accidents rarely occur . . . ,"[20] while Burrell claims that, "It is possible to avoid piercing the visceral pleura by using a blunt Riviere needle for the initial operation . . . ."[21] Other workers have similarly advocated a dull needle.[22-26]

In an attempt to clarify this controversial point, we recently studied 29 patients who were submitted for the induction of initial pneumothorax. In order to eliminate the sharp needle hazard, only dull needles were used (No. 19, short-beveled). As in the previous experimental study, the needle was introduced with the greatest care until pleural readings were obtained. At this point, the needle was withdrawn in 19 cases without giving any air whatsoever and in the remaining 10 cases approximately 100 cc. of air was given. *In each of the cases in which pleural readings were obtained a progressively increasing pneumothorax space was demonstrated by fluoroscopy and x-ray.* (In two cases, a pneumothorax space could not be clearly seen until pleural readings were obtained a second time). These studies have been reported elsewhere in detail.[27]

From these experiments, it was obvious that the mere taking of pleural readings (the first step in the routine technique of induction of pneumothorax) will produce a pneumothorax space, whether a sharp or dull needle be used. The gas contained in this pneumothorax space comes from the underlying lung. This is shown by the following facts: 1) Even when no air is introduced into the pleural space during the taking of readings, a pneumothorax space is almost always created. 2) When air is given, the collapse is always greater than might be expected from the amount introduced. 3) The pneumothorax space usually increases progressively over a period of hours. 4) Analysis of the gas that is in the pneumothorax space shows it to be alveolar air. *Thus, every initial pneumothorax is first a traumatic pneumothorax—in every successful initial pneumothorax the space is obtained after puncturing the underlying lung.*

Many facts difficult to explain otherwise become clear when the above findings are kept in mind:

1. The difficulty in obtaining a pneumothorax space in atelectatic lungs.
2. The unusually large collapse so frequently observed in cases of emphysema.
3. The greater frequency of air embolism during initial pneumothorax.

4. Instances in which readings cannot be obtained, nor air given, and the attempt considered unsuccessful, but which result in a good pneumothorax space on examination hours later.
5. Excessive collapse despite introduction of small amounts of air.

The recognition that injury to the lung is inevitable during induction of pneumothorax raises the question of whether or not it may be possible to minimize or regulate the trauma produced. That this is not an academic problem is emphasized by the occasional disasters following initial pneumothorax. Harris, in 1915, reported a death following initial pneumothorax, the patient dying 17 hours after induction, with massive collapse.[28] In the following decades, other deaths were reported.[10,13,14,26] In several of these cases, autopsy was performed and in each, rents were present in the lung. But far more frequent than cases ending fatally, are those instances of extensive collapse after initial pneumothorax, in which only immediate deflation has prevented death. The present study, reported herein, was prompted by a desire to devise means of minimizing trauma during induction of initial pneumothorax and thus avoiding dangerous, excessive pulmonary collapse.

Materials and Methods

Eighty-two consecutive patients recommended for pneumothorax therapy at Sea View Hospital were studied. The patients were divided into two groups. In the first group were 36 patients in whom induction was performed with a No. 19 dull, short-beveled needle. The induction varied from the usual procedure insofar as *little or no air was given* after pleural readings were taken. If symptoms suggestive of pneumothorax became evident (we found pain referred to the homolateral side of the neck or to the shoulder a reliable indication) the patient was immediately fluoroscoped and if a pneumothorax space was seen, a roentgenogram of the lung, in expiration, was taken at once. Subsequent roentgenograms were taken 3 hours later and the next day. If there were no symptoms "expiration" roentgenograms were routinely taken 3 hours and 24 hours after the pleural readings were obtained. We found early in our work that a relatively "soft" expiration x-ray was more reliable than fluoroscopy. In doubtful cases, therefore, the roentgenogram rather than the fluoroscopic appearance determined the presence and size of a pneumothorax space.

In the second group were 46 patients. In these cases induction was performed with a small needle, using a technique described elsewhere in detail.[29] Briefly, a No. 24 or 25 needle, attached to

a novocaine-filled syringe, was inserted through the chest wall and as soon as air-bubbles were aspirated, the needle was withdrawn, no air being given. Each patient was then studied in the manner described above.

From the data gathered on fluoroscopy and roentgenograms, an evaluation of the amount of the collapse produced was attempted. This is acknowledged to be arbitrary but since the same criteria were used in all cases, comparative estimations may be considered valid. In 15 patients (7 in group I and 8 in group II) the pleurae were adherent and no pneumothorax space could be obtained despite many attempts. The remaining 29 patients in group I and 38 patients in group II, a total of 67 patients, will be considered consecutive for the purposes of this study.

Results

The results of our investigations indicate that while lung perforation caused by the pneumothorax needle is the basic mechanism of collapse at induction, the degree of collapse is conditioned by the size of the needle used for the initial pneumothorax and other factors discussed below.

A. *Initial Pneumothorax in Unilateral Disease.*

Table I shows that the amount of collapse obtained in cases with unilateral disease with minimal or moderate lung involvement, was small. The amount of collapse was nearly twice as great if a large needle was used: in 15 patients in whom pneumothorax was induced with a small needle, the average collapse obtained was approximately 8 per cent while in 11 patients in whom a large needle was used, an average collapse of 15 per cent resulted. However, in none of the cases in this group was the collapse sufficient to produce dyspnoea.

In patients in whom the disease was extensive the difference in the collapse obtained by large and small needles was much more apparent. There were 11 cases of massive unilateral disease. In 7, pneumothorax was induced with a small-gauge needle—the average collapse was 8 per cent and in no case was there dyspnoea. In 4 comparable cases a large needle was used and pleural readings taken; the average collapse was 41 per cent and in 3 of these 4 cases, the patient complained of dyspnoea.

As can be seen from our experiences with patients in whom the disease is unilateral, the collapse will be greater if a large needle is used. This difference will be much more evident if the disease is extensive. It is thus important, in managing cases with massive unilateral involvement, to anticipate a certain degree of dyspnoea if a large needle is used for the initial pneumothorax.

TABLE I: Initial Pneumothorax in Unilateral Tuberculosis

Pulmonary Collapse With Large Needle	Case	Manometric Readings		Air Introduced	Maximum Collapse Per cent	Dyspnoea	Deflation
A. Minimal to moderate unilateral disease	1	Unsatisfactory		None	10	....	....
	2	−9	−6	None	8	....	....
	3	−5	−1	None	15	....	....
	4	Unsatisfactory		None	35	....	....
	5	−14	−8	100 cc.	10	....	..
	6	−12	−8	100 cc.	5	....	....
	7	−9	−2	None	15	....	
	8	−9	−6	None	20	....	....
	9	−10	−6	None	5	....	..
	10	−20	−15	None	5	....	....
	11	−10	−4	None	35	....	....
		Average collapse: 15%.				No instance of dyspnoea.	
B. Extensive unilateral disease	1	−16	−8	None	25	Moderate	..
	2	−14	−8	150 cc.	70	Minimal	....
	3	Unsatisfactory		None	35	Minimal	....
	4	−11	−8	None	35	....	....
		Average collapse: 41%.				Three instances of dyspnoea.	

Pulmonary Collapse With Small Needle							
A. Minimal to moderate unilateral disease	1	....	....	None	20	....	....
	2	....	....	None	10	....	....
	3	....	....	None	5	....	....
	4	....	....	None	5	....	....
	5	....	....	None	5	....	....
	6	....	....	None	5	....	....
	7	....	....	None	10	....	....
	8	....	....	None	5	....	....
	9	....	....	None	15	....	....
	10	....	....	None	5	....	....
	11	....	....	None	5	....	....
	12	....	....	None	5	....	....
	13	....	....	None	5	....	....
	14	....	....	None	10	....	....
	15	....	....	None	10	....	....
				Average collapse: 8%.	No instance of dyspnoea.		
B. Extensive unilateral disease	1	....	....	None	5	....	....
	2	....	....	None	8	....	....
	3	....	....	None	5	....	....
	4	....	....	None	5	....	....
	5	....	....	None	20	....	....
	6	....	....	None	5	....	....
	7	....	....	None	7	....	....
				Average collapse: 8%.	No instance of dyspnoea.		

TABLE II: Initial Pneumothorax in Bilateral Tuberculosis

Pulmonary Collapse With Large Needle	Case	Manometric Readings		Air Introduced	Maximum Collapse Per cent	Dyspnoea	Deflation
A. Each lung only moderately involved	1	−18	−6	None	15	....	....
	2	−12	−8	None	5	....	....
	3	−12	−8	None	10	....	....
	4	Unsatisfactory		None	35	Minimal	....
				Average collapse: 16%.		One instance of dyspnoea.	
B. Homolateral - moderate Contralateral - extensive	1	−7	−3	None	60	Marked	Intermittent
	2	−8	−2	None	20	Slight	....
	3	−10	?	None	90	Marked	Continuous
	4	−6	−4	200 cc.	50	Moderate	....
	5	−11	−4	150 cc.	80	Moderate	....
	6	−10	−8	25 cc.	10	....	....
	7	−20	−14	100 cc.	40	....	....
	8	−20	−3	100 cc.	50	Moderate	Intermittent
				Average collapse: 50%.		Six instances of dyspnoea.	
C. Both lungs extensively involved	1	−8	−6	150 cc.	45	Marked	Intermittent
	2	−6	?	100 cc.	50	....	....
				Average collapse: 47%.		One instance of dyspnoea.	

Pulmonary Collapse With Small Needle							
A. Each lung only moderately involved	1	....	....	None	5	....	....
	2	....	....	None	5	....	....
	3	....	....	None	5	....	....
	4	....	....	None	5	....	....
	5	....	....	None	5	....	....
	6	....	....	None	5	....	....
				Average collapse: 5%.	No instance of dyspnoea.		
B. Homolateral - moderate Contralateral - extensive	1	....	....	None	20	....	....
	2	....	....	None	20	....	....
	3	....	....	None	25	....	....
	4	....	....	None	40	Moderate	....
	5	....	....	None	35	....	....
	6	....	....	None	20	....	....
				Average collapse: 27%.	One instance of dyspnoea.		
C. Both lungs extensively involved	1	....	....	None	8	....	....
	2	....	....	None	75	Minimal	....
	3	....	....	None	20	....	....
	4	....	....	None	80	Marked	Intermittent
				Average collapse: 47%.	Two instances of dyspnoea.		

B. *Initial Pneumothorax in Bilateral Disease.*

In bilateral disease the use of the large needle caused dyspnoea and necessitated deflation far more frequently than was the case when a small needle was used. It can be seen from Table II that both the size of the needle and the status of the underlying lung are the major factors in determining the extent of the collapse obtained.

In cases in which the lung to be collapsed is only moderately diseased and in which the contralateral lung is also but moderately involved, the collapse obtained is usually limited. But here, too, needle size influences the extent of the collapse. In 6 bilateral cases, moderate in extent on each side, the collapse obtained with a small needle averaged 5 per cent. In no case did dyspnoea occur. In contrast, in 4 cases in which a large needle was used an average collapse of 16 per cent was obtained and in one case, slight dyspnoea developed.

This difference between the large-gauge and small-gauge needle becomes still more significant in cases in which the contralateral disease is extensive and the lung to be collapsed is only moderately involved. We induced pneumothorax in 6 such cases with a small-gauge needle. The average collapse was 27 per cent and in only one case did dyspnoea develop; it was moderate in nature and did not require deflation. On the other hand, in 8 comparable cases (homolateral moderate, contralateral extensive) the use of the large-gauge needle resulted in an average collapse of 50 per cent, double that of the small needle. Moreover, 6 of the 8 patients became dyspnoeic and 3 of these were so severely dyspnoeic as to

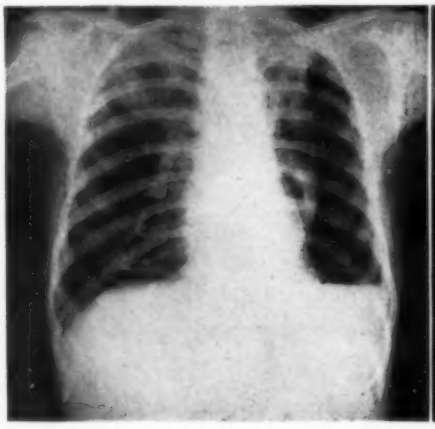

FIGURE 1

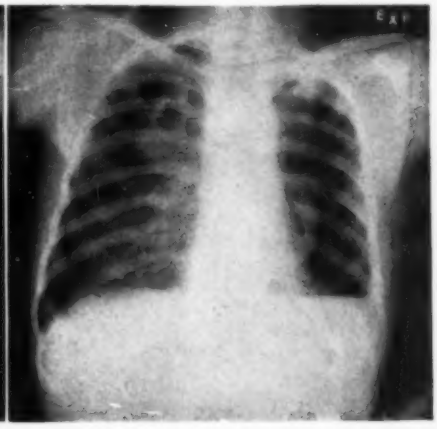

FIGURE 2

Figure 1, Case R.S.: Bilateral disease, with extensive exclusion of the contralateral lung by hydropneumothorax.—*Figure 2, Case R.S.:* Roentgenogram 4 hours after the induction of pneumothorax with a small-gauge needle (no air given). Collapse never exceeded 30 per cent and dyspnoea did not occur.

require deflation. Figures 1 - 4 illustrate typical experiences in this group, demonstrating the contrasting degrees of collapse obtained with the large and the small needle.

Finally, in cases in which the disease is extensive in both lungs, induction by any needle is probably dangerous. In our hands, both large and small needles resulted in marked collapse, averaging 50 per cent, and two of the cases we studied required deflation.

Discussion

An analysis of our results indicates that there are two principal factors determining the extent of the collapse in initial pneumothorax—the size of the needle used and the condition of the underlying lung. Since every initial pneumothorax is first a traumatic pneumothorax, it may be expected that a small-gauge needle will produce a smaller collapse, since the smaller the needle, the less the injury to the lung. Actually, our studies confirm this: on the overall picture, the use of the large-gauge needle will result in a collapse twice that found when a small-gauge needle is used.

In most cases with disease of moderate extent, it will make little difference, clinically, whether a small or large needle be used; the patient will rarely become dyspnoeic and will not require deflation. The situation is different in patients with extensive—especially bilateral—disease. These patients, because of their diminished respiratory reserve, are least able to withstand large

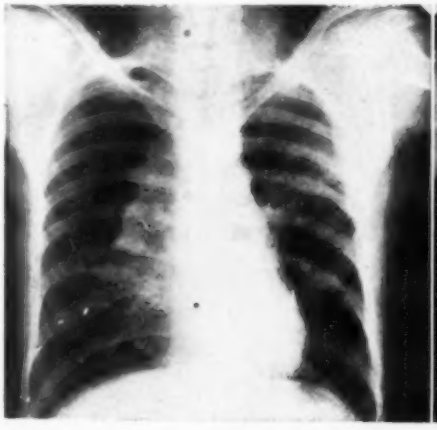

FIGURE 3

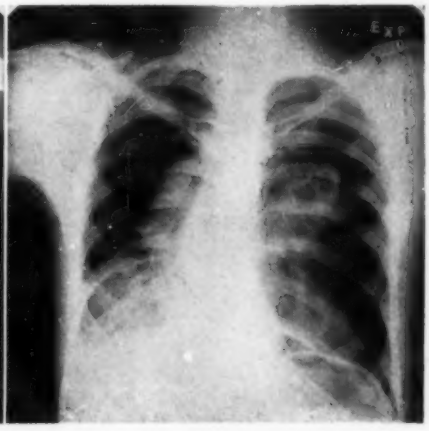

FIGURE 4

Figure 3, Case H.S.: Bilateral disease of similar extent and nature to that of Case R.S. in Figures 1 and 2.—*Figure 4:* In this patient, however, a large-gauge needle was used for the induction. After pleural readings of —20 on inspiration and —3 on expiration, 100 cc. of air was introduced. The resulting pneumothorax space showed progressive increase in size and at the time of this roentgenogram, 2 hours after initial pneumothorax, showed 30 per cent collapse. The size of the pneumothorax continued to increase and 24 hours after induction, with 50 per cent collapse, dyspnoea was complained of. Deflation became necessary.

collapse with its attendant diminution in vital capacity. Yet it is precisely in these cases that initial (traumatic) pneumothorax produces the largest collapse. This is true with the small-gauge needle as well as with the large-gauge. However, here the *relatively* smaller collapse obtained with the small-gauge needle becomes clinically important. This is amply illustrated by our experience in this series. We found that dyspnoea was much more common among cases in which induction was done with the large-gauge needle. In 29 cases of induction with the large-gauge needle, there occurred 11 (38 per cent) cases of dyspnoea of which 4 required deflation. In 38 cases in which a small-gauge needle was used, only 3 (8 per cent) had dyspnoea and only one of these required deflation. As can be seen from Tables I and II, dyspnoea occurred mostly in cases with bilateral disease.

Thus it is evident that in cases in which the disease is extensive, it is *almost mandatory, if complications are to be avoided, that a small-gauge needle be used for the initial pneumothorax.* Moreover, even if the disease is not extensive but if the vital capacity is impaired for any reason (severe emphysema, cardiac disease, scoliosis, etc.) it is preferable to use the small-gauge needle in order to obtain a moderate collapse. Figures 5 - 6 illustrate a deliberate therapeutic application of this principle.

Just as important as needle-size is the condition of the underlying lung. In bilateral cases, as noted above, collapse is twice that found in unilateral disease. And even in unilateral disease

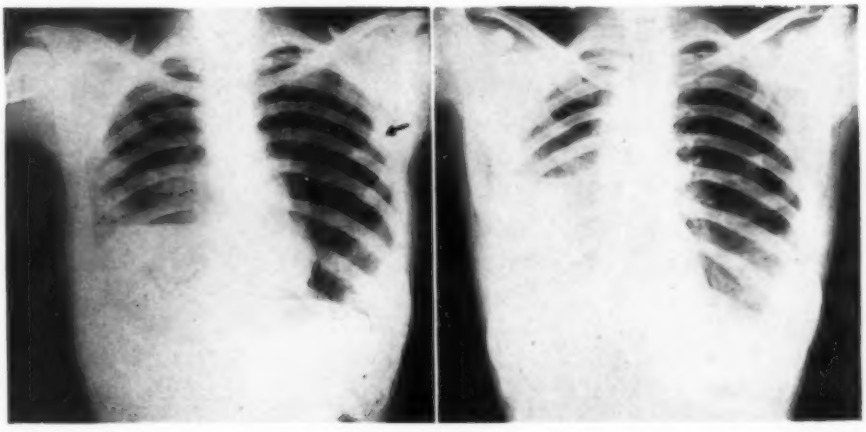

FIGURE 5 FIGURE 6

Figure 5, Case A.H.: Pneumothorax to be induced on the left, in the face of exclusion of the right lung by hydropneumothorax. Because of the special dangers in this type of case, a small needle was recommended for the initial pneumothorax.—*Figure 6, Case A.H.:* Roentgenogram 4 hours after the induction of pneumothorax with a small-gauge needle (no air given). This collapse of approximately 20 per cent was the maximum obtained and the patient remained comfortable, with no dyspnoea.

(when the large needle is used) the collapse is much greater when the disease is extensive rather than moderate.

An analysis of our cases indicates that it was the extent, rather than the character, of the pulmonary disease which was of importance. It apparently made little difference what the process was which excluded a portion of the lung from respiratory function: caseous infiltration, pulmonary fibrosis, contralateral pneumothorax or thoracoplasty, pleural thickening, hydropneumothorax—each was of significance only insofar as it destroyed or excluded functioning pulmonary parenchyma. It seems to us that the most plausible explanation of this phenomenon lies in the fact that in lungs extensively involved, the remaining functioning parenchyma is constantly active, compensating for the non-functioning areas. Unfortunately, it is precisely these uninvolved areas, distended by compensatory emphysema, constantly active, which we have to choose for the induction of pneumothorax, in order to avoid pleural infection from areas of the lung involved by tuberculosis. And with trauma inevitable during the initial pneumothorax, there is a greater escape of alveolar air, since the emphysematous lung retracts poorly and *the perforation* is kept open by the increased activity of the area. These two circumstances—the altered condition of the remaining functioning parenchyma plus the trauma of the initial pneumothorax—combine to make induction of pneumothorax dangerous in the presence of extensive disease. And since we cannot alter the condition of the lung parenchyma, there remains only the possibility of minimizing the inevitable trauma through the use of the small-gauge needle.

SUMMARY AND CONCLUSIONS

1) *Every* initial pneumothorax is of necessity first a traumatic pneumothorax. The needle inserted into the (potential) pleural space punctures the visceral pleura and air escapes into the pleural space from the lung. This is true irrespective of the type or character of the needle used.

2) Initial manometer readings are those of this traumatically produced pneumothorax; it is into this space that we add air from the pneumothorax apparatus.

3) The trauma to the lung varies with the size of the needle used. Small needles produce small openings into the lung, less air escapes.

4) Several additional factors determine the final size of the pneumothorax. Most important are the existence of contralateral involvement and the status of the underlying lung. If the disease is extensive, the remaining functioning parenchyma shows compensatory emphysema. When such lung is injured, air leakage

is persistent and is aggravated by the constant activity of this functioning part of the lung. Severe and even dangerous dyspnoea may result.

5) Of the two principal factors determining the extent of the traumatic pneumothorax—the condition of the lungs and the size of the needle used—only the latter can be varied. The smaller needle, resulting in less trauma, gives a smaller collapse. In sixty-seven initial pneumothoraces, the collapse obtained with a large (No. 19) needle was approximately twice that noted with a small needle.

6) With moderate pulmonary involvement the size of the needle is of minor importance since excessive collapse and dyspnoea are uncommon.

7) However, in extensive disease the use of a large needle frequently results in marked collapse, often accompanied by severe dyspnoea, requiring deflation. In 16 bilateral cases in which pneumothorax was induced with a small needle, 3 became dyspnoeic, 1 requiring deflation. In 14 similar cases induced with a large needle, 8 became dyspnoeic and 4 of these required deflation.

In patients with extensive disease, the dangers of induction of the initial pneumothorax may be minimized by the use of a small-gauge needle. In bilateral cases, its use appears essential if a manageable collapse is to be obtained.

RESUMEN Y CONCLUSIONES

1) Todo neumotórax inicial es necesariamente un neumotórax traumático al principio. La aguja que se introduce en el espacio pleural (potencial) agujerea la pleura visceral y permite el escape de aire del pulmón al espacio pleural. Sucede esto no importa cual sea el tipo o la clase de aguja que se emplee.

2) Las indicaciones iniciales del manómetro se deben al neumotórax producido por este traumatismo, y es a este espacio al que añadimos aire mediante el aparato de neumotórax.

3) El traumatismo causado al pulmón varía de acuerdo con el tamaño de la aguja empleada. Agujas pequeñas producen agujeros pequeños en el pulmón y menos aire se escapa.

4) Varios factores adicionales determinan el tamaño final del neumotórax. Los más importantes son la existencia de lesiones contralaterales y el estado del pulmón subyacente. Si la enfermedad es extensa, el parénquima funcional restante sufre un enfisema compensatorio. Cuando se hiere a tal pulmón, el escape de aire es persistente y lo agrava la actividad constante de esta parte funcional del pulmón. Puede resultar disnea grave y hasta peligrosa.

5) De los dos factores principales que determinan el tamaño

del neumotórax traumático—la condición de los pulmones y el tamaño de la aguja empleada—sólo el último puede ser variado. Las agujas más pequeñas causan menos traumatismo y, por consiguiente, ocasionan colapsos más pequeños. En sesenta y siete neumotórax iniciales, el colapso obtenido con una aguja grande (No. 19) fue aproximadamente el doble del que se notó con una aguja pequeña.

6) Con una lesión pulmonar moderada el tamaño de la aguja es de poca importancia, ya que demasiado colapso y disnea son raros en estos casos.

7) Sin embargo, cuando la enfermedad es extensa el empleo de una aguja grande resulta frecuentemente en marcado colapso, acompañado a menudo de grave disnea que requiere desinflación. En 16 casos bilaterales en los que se inició el neumotórax con una aguja pequeña, 3 tuvieron disnea y hubo que desinflar a uno. En 14 casos semejantes iniciados con una aguja grande, 8 tuvieron disnea y fue necesario desinflar a 4.

En pacientes con lesiones extensas, es posible reducir al mínimo los peligros del neumotórax inicial mediante el uso de una aguja pequeña. Su empleo parece ser esencial en casos bilaterales si se desea obtener un colapso manejable.

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Indications for Pulmonary Resection for Tuberculosis Both by Lobectomy and Pneumonectomy*

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INTRODUCTION

The actual role of pulmonary resection in the treatment of tuberculosis is one of the most disputed questions in the surgical therapy of this disease. With the great improvements in the surgical management of lobectomy and pneumonectomy, together with a better understanding of the physiology of the thoracic organs, and the use of powerful antibiotics, surgeons are encouraged as never before to strike directly at the problem of extirpating the diseased portions of the lung. Yet despite these advances, pulmonary resection still remains a formidable procedure, one not to be lightly recommended. The authors believe that pulmonary resection should not be advised in competition to the standard collapse measures used in the treatment of pulmonary tuberculosis, but that resection is to be performed when these measures have failed or obviously will not succeed. The purpose of this discussion is to present a practical consideration of the problem, based on the management of over a thousand cases of pulmonary tuberculosis by various surgical measures during the recent years.

General Considerations

With the present day surgical technique, the operative results in pulmonary resection for pulmonary tuberculosis are far superior to those of even a few years ago. The important points in these improved methods are discussed later, under "Surgical Technique." Yet, in reviewing our cases and the reports in the recent literature, we are impressed with the fact that following pulmonary resection for tuberculosis, the percentage of cures where the patient has a negative sputum on culture varies from 40 to 45 per cent while the overall mortality ranges from 14 to 38 per cent. If we compare these results with those following thoracoplasty, which is the procedure that has given the best results when medical measures including pneumothorax have failed, a striking contrast is seen.

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The present day postoperative mortality for thoracoplasty is less than 2 per cent and from 65 to 80 per cent cures are reported in the literature from many clinics. It is apparent, therefore, despite the great strides that have been made in reducing the serious complications in pulmonary resection, that the following considerations concerning resection must be carefully weighed:

1) The mortality rate and morbidity are much higher following resection than following thoracoplasty.

2) Contralateral and ipsolateral spreads or re-inactivations are much more frequent.

3) Bronchial fistula and empyema are much more frequent.

4) If empyema develops or a tuberculous pleural effusion persists for some time, the remaining lung beneath will be to a greater or lesser degree immobilized with loss of function. Even though no such complication develops, some reduction in function occurs because of the adherence which usually takes place between the chest wall and the remaining lobe or lobes.

5) Less frequently, than by thoracoplasty, does the sputum or material obtained by gastric lavage become free of tubercle bacilli and remain so following lobectomy and pneumonectomy.

Without doubt, lung resection is a valuable adjunct in our therapeutic contest against pulmonary tuberculosis. It will increase in its worth as technical difficulties are overcome, however we are still of the opinion that thoracoplasty is to be preferred for many of the cases reported in the literature, where lung resection is now being employed.

Pulmonary tuberculosis is essentially a bilateral disease. The aim in combating it is to preserve as much pulmonary tissue as possible, contingent upon the arrest of the tuberculous process. Accordingly, when applicable, "reversible" immobilizing or collapsing measures are employed in preference to "irreversible" ones. These "reversible" measures are intrapleural pneumothorax, pneumonolysis, temporary diaphragmatic paralysis, pneumoperitoneum, and extrapleural pneumothorax. If, after some, but not prolonged observation, these procedures are not successful, or are not justified, then "irreversible" procedures are considered. When thoracoplasty probably, even possibly, may be followed by sputum conversion, we feel it is indicated rather than lobe or lung resection. The thoracoplasty must affect the maximum collapse of the lung possible, and should include complete removal of the upper ribs, transverse processes plus a "lysis" of the lung, when indicated. The collapse should be continued by the removal of a sufficient number of ribs to insure effective closure of the pulmonary cavities. Thoracoplasty, if unsuccessful, may then be followed by removal of the offending pulmonary tissue.

Indications for Pulmonary Resection

Our main indication for pulmonary resection is the failure of thoracoplasty to control the disease. If, following thoracoplasty, a residual cavity is demonstrated by Potter-Bucky films or planograms to be the source of the tubercle bacilli in the sputum, then some form of surgery must be considered. However, before deciding upon pulmonary resection, the following possibilities should be eliminated:

1) Revision thoracoplasty.
2) The formation of an extrapleural space and possible insertion of some sort of pack, or plombage.
3) Cavity drainage.
4) In some cases, no further surgery.

If revision thoracoplasty may increase the collapse of involved lung sufficiently to obliterate a nearly closed cavity and thus affect sputum conversion, it certainly should be tried. Revision thoracoplasty is most often the procedure of choice when thoracoplasty has failed. If it is performed in graded stages, the mortality is low. If there is no absolute contra-indication, such as a bronchial stenosis, the results are most encouraging.

The development of an intra or extrapleural space directly over the residual cavity, with or without an apicolysis, followed by the introduction of wax or some other form of pack has been carried out bu us in more than 30 cases. Approximately 50 per cent were successful in converting sputum negative to culture.

External drainage of a residual cavity after an unsuccessful thoracoplasty, maintaining drainage by means of a pedicled skin flap has also resulted in about 50 per cent apparent cures. However, revision thoracoplasty is preferred to either plombage or cavity drainage. The failures of the latter are very disheartening to both patients and doctors. Success is assured only after weeks or months of prolonged hospitalization, frequent dressings and discomfort to the patient.

If the remaining cavity is small and if the sputum is positive, only occasionally by culture; if the patient is intelligent and responsible; in certain instances it is permissible, in our opinion, to advise against further surgery with the definite understanding that such a person check regularly with a physician skilled in recognizing premonitory or early signs of trouble.

Thoracoplasty fails primarily as a result of four main factors: namely, tension cavities, endobronchial disease, cavities located close to the spine and bronchiectatic cavities.

1) *Tension cavities:* The behavior of a tuberculous lung cavity is unpredictable. Its persistence depends in a very large measure

on the relationship of the cavity to its bronchus or bronchi. Because of viscid or inspissated secretions, or necrosing tissue in the cavity at the bronchial opening or within the bronchus itself; because of bronchial disease either ulcerative or cicatricial; because of edema of the bronchial walls and local or widespread bronchial spasm; because of softening and kinking of bronchial walls proximal to the cavity with which it is joined, a check-valve mechanism may develop in that bronchus or at the broncho-cavitary junction.

The enlargement of the bronchial lumen that occurs in a normal bronchus during inspiration, probably takes place in some degree in an edematous one especially during the act of coughing. With expiration, as the bronchial lumen narrows, some air may be trapped. As this phenomenon continues, a tension cavity develops. Undoubtedly other factors lead to the accumulation of air within tuberculous cavities producing positive pressure within them.

Unfortunately, we cannot determine the cause in any given case of the check-valve mechanism. Similarly, we are unable to anticipate what may lead to its elimination. Under no treatment whatsoever, a cavity may increase or decrease astonishingly in size in a matter of days or even hours. With pneumothorax, a cavity may increase 100 per cent or more in size as compared with the condition before pneumothorax was instituted. It may at once disappear when the pneumothorax has been abandoned and re-expansion of the lung takes place.

Immediately following "phrenic crush," a large tension cavity may disappear perhaps to reopen when the diaphragmatic leaf returns to active motion. With the so-called "Monaldi drainage," a cavity may seem to be closed, only to return when the catheter draining the cavity is extruded.

And so it is with thoracoplasty. One can never be sure except by trial whether a tension cavity, perhaps a large one, will disappear with sputum conversion. Nor can we be confident that a small thin-walled cavity near the periphery (where prognosis for closure by thoracoplasty should be excellent) will not persist, even increase in size, during the course of the thoracoplastic stages. Statements to patients concerning thoracoplastic results must therefore be guarded for we are unable to predict broncho-cavity behavior.

Throughout all these weeks or months of treatment, success has depended upon the production of an advantageous alteration in the broncho-cavitary relationship that would afford a constantly adequate communication between the cavity or cavities under treatment and their bronchi. Since we cannot as yet anticipate when, if at all, such a desirable relationship may take place, we advise the much less serious thoracoplasty before re-

commending lobectomy. It should be remembered that at operation, pathologic changes may be found that make the proposed lobectomy not feasible and necessitate a complete removal of the lung. Thus lobectomy or pneumonectomy are indicated when thoracoplasty is unsuccessful in closing a tension cavity (Case 1).

2) *Endobronchial disease:* Endobronchial tuberculosis is not necessarily an indication for lobe or lung removal. The mild forms of tuberculous bronchitis showing only hyperemia and edema of the bronchial mucosa in no way change the general indiaction for treatment. Similarly, a submucosal tuberculous lesion which is more of a pathologic than a clinical diagnosis, does not constitute an indication for pulmonary extirpation. Many surgeons consider tuberculous ulceration an indication for lobectomy or pneumonectomy, as the prognosis with thoracoplasty in this type lesion was not good. Up until the past year, we have treated these lesions with cauterization of silver nitrate and proceeded with the thoracoplasty. Frequently, the resultant cicatricial stenosis of the

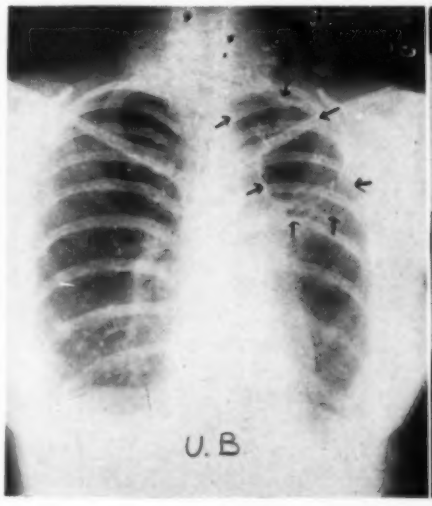

FIGURE 1A

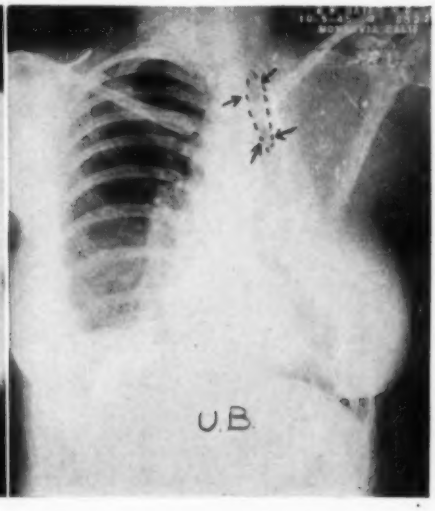

FIGURE 1B

CASE 1: This 23 year old female gave a history of cough productive of large amounts of sputum, fever and chronic fatigue of two years duration. Pneumothorax had been tried unsuccessfully. The patient was toxic and dyspnoeic with a daily fever of 102 degrees. Figure 1A shows a huge tension cavity of the left upper lobe of the lung with scattered foci on the right. Because the patient had no demonstrable bronchostenosis by bronchoscopic examination and because of the instability of the pulmonary lesion, a thoracoplasty was performed. Figure 1B shows persistent cavity of the upper lobe of the left lung following thoracoplasty, and a revision of the thoracoplasty. The right sided pulmonary lesion was now stable enough to permit lobectomy of the left upper lobe. The patient has been well for over one year following this procedure. This case was not a suitable risk for lobectomy when the thoracoplasty was performed. The decrease in fever, toxicity and the amount of sputum along with the stabilization of the right sided lesion made the patient a satisfactory subject for pulmonary resection.

bronchus necessitated ultimate lung removal. However, the use of streptomycin for the treatment of tuberculous ulcers of the bronchus, as described later in this paper, seems to be the treatment of choice.

Cicatricial stenosis of the bronchus which results from a tuberculous tracheal bronchitis, is a very important indication for pulmonary resection. However, with gentle dilation of the narrowed portion of the bronchus, drainage may be established so that the thoracoplasty may be successful in closing the pulmonary cavity. In some instances, adequate collapse of the pulmonary cavities and ultimate conversion of the sputum, was effected even though drainage from the bronchus was not well established before the thoracoplasty was undertaken. Since the operative risk is far less, and since the spread to the contralateral lung is unusual with thoracoplasty, we believe that this surgical procedure

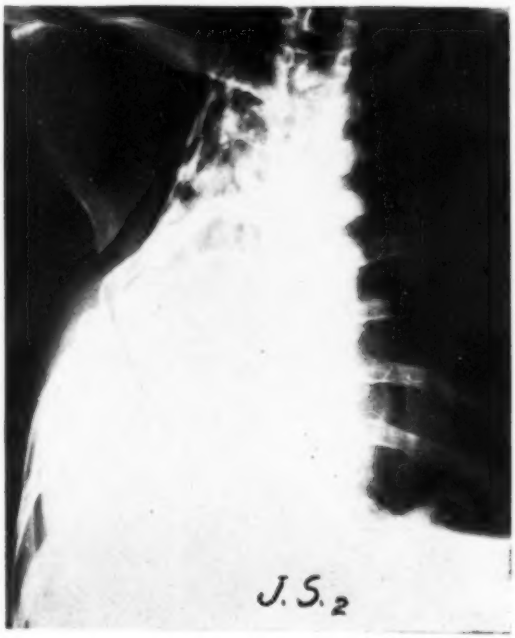

FIGURE 2

CASE 2: A male 33 years old was treated for pulmonary tuberculosis for ten years. There had been cyclic attacks of productive cough and fever during this period of time. Pneumothorax and oleothorax had been unsuccessful in controlling the right sided pulmonary lesion. Bronchoscopy revealed a tuberculous fibrous stenosis of the right upper lobe branch bronchus. Drainage of the right upper lobe was effected by bronchoscopic dilations of the stenosis and thoracoplasty resulted in apparent control of the upper lobe disease. However, one year later the symptoms of bronchial obstruction again occurred and bronchoscopy showed that the right main bronchus was now almost completely obstructed with a fibrous stenosis. The x-ray now showed cavitation and atelectasis of the entire right lung (Fig. 2). Because of progressive bronchostenosis, thoracoplasty had failed and pneumonectomy was performed. The patient is now well.

should be given a fair trial before attempting pulmonary resection. If in a case with bronchial stenosis, complete collapse of the lung is obtained with intrapleural pneumothorax but the sputum remains positive, then it is manifest that a thoracoplasty will secure no greater collapse and a "primary pneumonectomy" is indicated. If thoracoplasty has been tried and has failed, then pulmonary resection is the procedure of choice (Case 2).

3) *When a cavity lies close to the mediastinum and hilum:* Such a cavity, so disadvantageously located, has seldom been closed by either intrapleural pneumothorax or extrapleural pneumothorax. We do not feel warranted, however, in recommending primary lobectomy under such conditions since it is by no means rare to obtain cavity obliteration and sputum conversion by thoracoplasty alone.

If, however, the cavity is thin-walled, especially if recent in development, even though it be located close to the mediastinum near the hilum, its closure may follow either of the reversible procedures first mentioned. If these are not successful, thoracoplasty often is followed by sputum conversion.

Lobe removal, then, is indicated if no other measure has availed (Case 3). It should again be emphasized that at operation it is often far better to abandon the attempt to remove a lobe if the presence of disease in the adjoining lobe (only discovered at operation) precludes the excision of all tuberculosis except by removal of the entire lung.

4) *Bronchiectasis:* Thoracoplasty in a patient with pulmonary tuberculosis may fail to control a recurrent hemoptysis. Frequently the cause of the hemorrhage is found in bronchiectatic cavitation. In some instances it is extremely difficult to ascertain the exact site of the hemorrhage, particularly if there is bilateral pulmonary fibrosis. Before considering resection, the surgeon must exercise great care to ascertain the exact source of the pulmonary bleeding. Some patients can tell exactly the site of a hemoptysis. Bronchograms are helpful if it is possible to fill the bronchiectatic dilatation responsible for the hemorrhage. However, if the bronchogram does not prove satisfactory, then a bronchoscopy must be performed when the patient is hemorrhaging so that the source of the hemorrhage can be accurately determined. If the findings at bronchoscopy are equivocal, then this procedure must be repeated until the surgeon is certain of the particular lobe that is involved. Then, even though the bronchograms and planograms have not been conclusive, lobectomy may be carried out. However, mild degrees of bronchiectasis and asymptomatic types do not constitute an indication for resection. Thus, the persistence of some cough and expectoration following thoracoplasty, and even an occasional

blood-streaking of the sputum do not demand removal of the offending pulmonary tissue.

Indications for "Primary Resection"

1) *Lower and middle lobe cavities:* If pneumothorax is not immediately successful in closing the cavity, it is not at once abandoned. Based on the premise described above, the persistence is likely to be due to inadequate bronchial drainage from the cavity or cavities present. We have found it worth while therefore, to change in as many ways as possible lines of stress and strain within the lung, hoping by so doing to facilitate the maintenance of a constant and sufficient bronchial opening from the cavity or cavities. Accordingly, instead of abandoning the pneumothorax pocket, temporary diaphragmatic paralysis is added. If this mechanical alteration has not an important effect on the cavity,

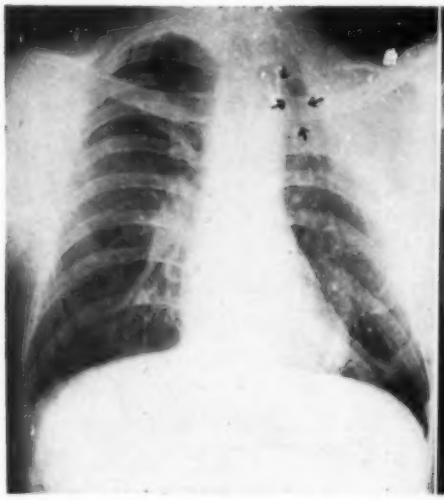

FIGURE 3

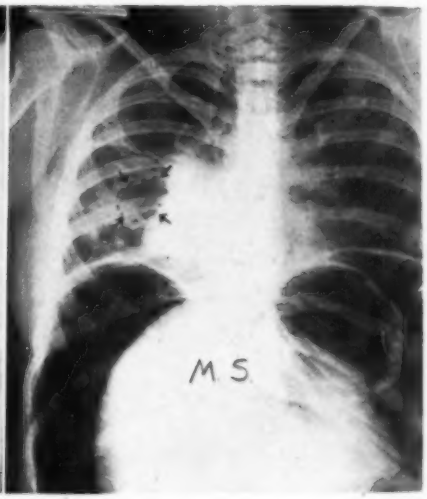

FIGURE 4

CASE 3: This 47 year old male had pulmonary tuberculosis for seven years during which time he had alternately worked and rested. Following an unsuccessful pneumothorax, a paraffine plombage was performed to collapse the cavity of the left upper lobe of the lung. The cavity remained open and a subsequent thoracoplasty after removal of the paraffine also failed to collapse the cavity. X-ray (Fig. 3) shows a persistent cavity close to the spine following thoracoplasty. Revision thoracoplasty seemed to be contraindicated because of the position of the cavity and a lobectomy was performed. The patient has been well for six years.

CASE 4: A 28 year old female had a productive cough with positive sputum for a period of three years. First a right pneumothorax and then a pneumoperitoneum with a temporary phrenic crushing were unsuccessful in closing a cavity of the right lower lobe. Figure 4 shows the cavity persisting in the right lower lung field following these procedures. Primary lobectomy of the right lower lobe was indicated, as there have been very poor results with thoracoplasty for lower lobe cavities. Thoracoplasty, which was not performed soon after the lobectomy, had to be subsequently done to control upper lobe disease.

pneumoperitoneum is instituted. If still no encouraging improvement follows, pneumothorax refills are stopped. If, after all these measures, closure is not obtained, we believe that removal of the lower lobe is indicated (Case 4).

2) *Blocked cavities and tuberculomata:* Experience has repeatedly demonstrated that expulsion of tuberculous material from a previously blocked cavity, often leading to spread of the disease to other lobes, is notoriously prone to occur. The presence of such a shadow, particularly if the patient complains of a mildly disturbing non-productive cough, constitutes a definite indication for lobectomy without a preceding trial thoracoplasty. There exists, of course, the possibility that the mass is carcinomatous in nature.

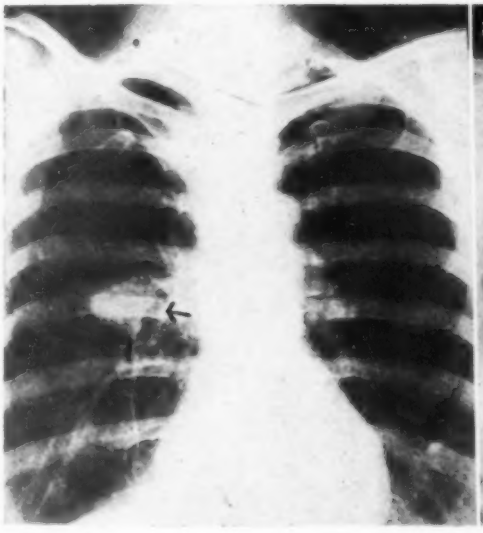

FIGURE 5

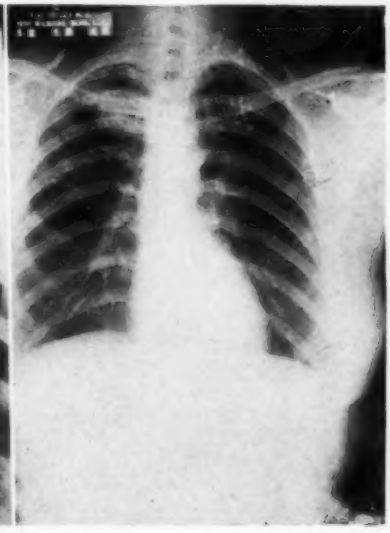

FIGURE 6

CASE 5: This 54 year old female complained of a slight cough and weakness of six months duration, along with some gastrointestinal complaints in the form of nausea, flatulence, and abdominal distress. The sputum was negative for acid fast bacilli on culture. After the production of a diagnostic pneumothorax, thoracoscopy showed no tubercles on the pleura and some sort of a mass in the upper lobe of the right lung. Because bronchogenic carcinoma could not be ruled out, the right upper lobe was resected. A tuberculoma was proven by pathological examination. The patient has been well for two years and this seems to vindicate the indication of resection of a pulmonary tuberculoma.

CASE 6: A female aged 41 years complained of a dry cough and fever of several weeks duration. Bronchoscopy showed no tumor and the sputum obtained by this procedure was negative for acid fast bacilli on culture. We advised removal of the right upper lobe of the lung, but the patient had few symptoms and refused surgery. Six months later the lesion broke down with a bronchogenic spread of the tuberculosis to both lower lobes of the lung. An extensive thoracoplasty was necessary to control the right sided disease. It seems likely that resection of the lobe of the lung containing this blocked cavity before the lesion had broken into the bronchial tree would have prevented the diffuse spread of infection to the other lobes. Figure 6 shows the shadow in the right upper lobe of the lung which remained static for a period of 6 months before breaking into the bronchus.

Whether tuberculous or carcinomatous therefore, the early removal of the lesion is indicated (Cases 5 and 6).

3) *Persistent bronchostenosis:* We have previously stated that if a cicatricial stenosis of the bronchus may be dilated successfully with bronchoscopic treatments, so that the resultant operation gives adequate drainage from the effected lobe or lobes, then thoracoplasty has a good chance of being successful. However, if it is impossible to secure a satisfactory airway by bronchoscopic dilations, then the thoracoplasty is doomed to failure. Often, for technical reasons, such as the angle at which an upper lobe bronchus comes off the main stem bronchus, it is impossible to adequately dilate a visible bronchial stenosis. In these instances where it is a foregone conclusion that thoracoplasty will not

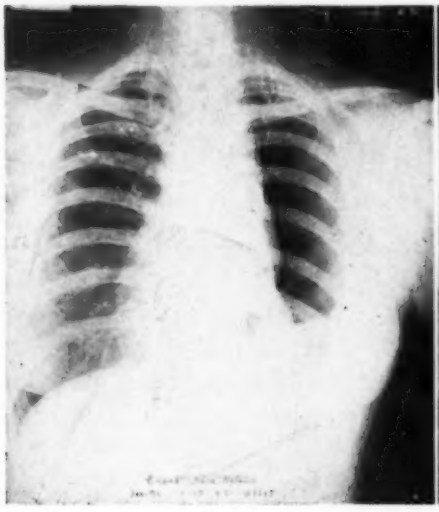

FIGURE 7

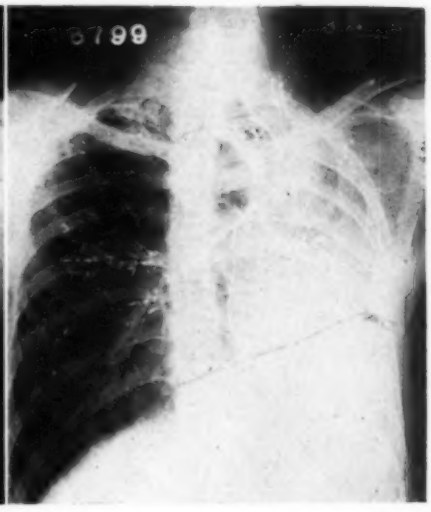

FIGURE 8

CASE 7: This 50 year old female had pulmonary tuberculosis diagnosed 23 years previously. After a year in the sanatorium she remained well for 17 years. Following a hemoptysis a pneumothorax was instituted on the left and although she rested for the next six years, the sputum remained positive for tubercle bacilli. X-ray (Fig. 7) demonstrated a "ground glass" appearance of the almost completely collapsed left lung. Because of the non-dilatable stenosis of the bronchus, proper drainage could not be established. This resulted in an "intrinsic atelectasis" of the lung, in which although the collapse of the lung was considerable (certainly more than could be expected by thoracoplasty) the sputum was strongly positive for acid fast bacilli on direct smear. Because no other form of therapy offered a reasonable chance of cure, primary pneumonectomy was indicated. She has remained well for 2½ years.

CASE 8: A male aged 49 years had suffered from pulmonary tuberculosis for 21 years. He had first been treated by rest, then a left sided pneumothorax which was discontinued because of the formation of fluid. For the 10 years preceding our treatment of the patient there had been increasing weakness, harassing cough and the raising of several ounces of purulent sputum daily. The sputum was intermittently positive. A bronchogram revealed total bronchiectasis of the left lung—with shift of the heart and mediastinum to the left. It was obvious that thoracoplasty would be ineffectual and a total pneumonectomy was performed. He has been well for over 1 year.

succeed, primary pulmonary resection is the procedure of choice. In other instances the stenosis is not visible with the bronchoscope but the bronchostenosis may be suspected by the homogeneous "ground glass" appearance of the lobe or lung in the x-ray. This is particularly apparent when a pneumothorax has been performed and this "ground glass" atelectasis of the lung is seen. We use the term "intrinsic atelectasis" for these cases to indicate its bronchial origin. In these cases, continuation of the pneumothorax is not likely to close the cavity; while abandoning the pneumothorax may be unsuccessful for the same reason; namely, because of the bronchostenosis. If high negative pressures are used, then fluid may develop, which is also undesirable. The expansion of the lung is desirable if it can be accomplished and cavity closure is secured during re-expansion in some cases. However, when the collapse of the lung is greater than that possible with a thoracoplasty and the sputum remains positive and when expansion is not readily obtainable, then primary pulmonary resection is the treatment of choice (Case 7).

4) *Bronchiectasis:* Bronchiectatic dilatations are frequently the aftermath of extensive tuberculous involvement of the pulmonary parenchyma. In these cases, a low grade tuberculous process may be present in the bronchiectatic sacculations and the sputum is often negative for acid fast bacilli except when an acute respiratory infection is present. Recurrent hemoptysis are common. Occasionally the tuberculous etiology of this condition may be overlooked in what appears to be a straightforward case of non-specific bronchiectasis, and the true nature of the pathology found only when microscopic sections are made of the resected pulmonary tissue following a lobectomy. A careful history, study of previous chest roentgenograms, and repeated sputum cultures will usually establish the tuberculous etiology. Primary pulmonary resection is indicated in these cases of bronchiectasis with definite symptoms or positive sputum, provided active pulmonary tuberculosis in the remaining lobes can be ruled out (Case 8).

Physiological Considerations

In studying any patient for either lobectomy or pneumonectomy, due regard must be paid to the cardiopulmonary reserve. If the patient will be made a respiratory cripple or go into pulmonary decompensation following resection of a lobe or lung, it is obvious that the operation is contra-indicated. The functional capacity of the contralateral lung and remaining lobe is, in most instances, the deciding factor. Bronchospirometry with differential oxygen consumption and blood gas analysis, though desirable, are generally not available and not yet standardized. We have relied on a

general clinical evaluation of the patient, together with simple breathing tests at rest or with mild exercise, the vital capacity and breath holding time. It is important to estimate the function of the remaining pulmonary tissue in the contralateral lung, in case of pneumonectomy and in the additional remaining lobes in a proposed lobectomy. With an intended lobectomy, it should be definitely decided before the operation whether or not the patient will stand a pneumonectomy, for lesions not seen on the chest roentgenogram are often found at operation which will make pneumonectomy necessary for the ultimate cure of the patient. Of course, if the lung is totally involved, then it can be safely assumed that the function of respiration is being carried on by the opposite lung.

The pulmonary function is made up of two main parts: (1) The action of the diaphragm and chest wall and (2) the capacity of the underlying lung to receive and exchange oxygen and CO_2. Diaphragmatic paralysis, often the result of an ill-advised permanent phrenic operation, or the immobility of the diaphragm from pleural adhesion, greatly embarrasses the functioning of the lungs. A previous pleurisy may prove most serious if it limits the movements of the chest wall. In this connection, the development of pleural fluid in any pneumothorax space must be considered as serious, if future optimum function of the underlying lung is needed. The physiological capacity of the lung is also dependent upon the amount of normal pulmonary tissue that is present. Thus, the degree of fibrosis, calcification and emphysema present in the lung, should be carefully evaluated. There are usually some of these changes present in most cases of pulmonary tuberculosis. Where pulmonary function is already reduced by some of these complications, any mediastinal shift, kinking of the trachea or main bronchus, or bronchial occlusion further impedes the ingress and egress of air to the lung.

Although the vital capacity is only a relative test of pulmonary function, any reduction below 1500 cc. for the average patient must be viewed with concern. Similarly, a breath holding time less than 15 seconds denotes a poor pulmonary reserve. Cardiac embarrassment, although detected by the usual tests, must be suspected if displacement of the mediastinum is marked. Amyloidosis, which may be diagnosed by the congo-red test, often produces marked alterations in the physiology of the patient. Care must be taken in the planning of the pre and postoperative treatment of these cases.

Low pulmonary capacity may contra-indicate a lobectomy, which otherwise would be the ideal therapeutic procedure. In these cases the surgeon is justified in attempting the other measures men-

tioned previously; namely, cavity drainage or the formation of an extrapleural space with the insertion of some sort of pack. These two procedures have less adverse effect on pulmonary reserve than revision thoracoplasty, and so can be used for the more desparate cases. Revision thoracoplasty, on the other hand, can be used for those that are not a suitable risk for resection.

Pathological Considerations

The type of pulmonary tuberculosis and the actual pathological changes in the lungs are of primary concern. For the most part, the reports in the literature have not accurately classified the cases according to the actual pathologic pulmonary findings. Yet the results must be evaluated in light of the pathologic pulmonary lesions if standardized criteria for pulmonary resections are to be established. The simplest classification embodies the character of the pulmonary lesion and the method of spread. It is important to consider whether or not the tuberculous process is predominently caseating or fibroid, and whether the disease is acute or chronic and spread through the bronchial tree. The stability of the pulmonary lesion, the broncho-cavitary relationship (as previously discussed), and amount and character of the sputum are important mitigating factors. Tuberculous lesions showing lymphogenous and hematogenous dissemination are usually bilateral and progressive. To expect a good late result from pulmonary resection in a case of pulmonary tuberculosis with protracted hematogenous dissemination, is out of the question. Up to the present time, the emphasis has been on the mere survival after the operation. However, with the lowered early mortality rate, attention should be turned to correlating the late results with the pathologic condition in the lungs.

Preparation of the Patient for Pulmonary Resection

Once the advisability for pulmonary resection has been decided upon, it behooves the surgeon not only to pick the proper time to perform the operation but also to get the patient in as satisfactory condition as possible to undergo surgery.

A. *Selection of time for performing operation:* In general, the patients with advancing pulmonary lesions are not fit subjects for lobectomy and pneumonectomy. Even with the aid of the powerful antibiotic, streptomycin, the lesion in the contralateral lung should be static or arrested. It is desirable that any lesion of the contralateral lung be quiescent for a period of at least a year before resection is carried out. We have performed lobectomy and pneumonectomy where the contralateral lesion had been inactive for less than a year with a recrudescence of these lesions.

All of the standard criteria for the determination of "activity" in tuberculosis should be used. The temperature, pulse and respiration, blood counts and sedimentation rates, if followed over a period of time, offer important clues as to the activity of any given lesion. Patients who are steadily losing weight, generally have progressive tuberculosis. Investigation of the gastro-intestinal or genito-urinary tract should be made if the symptoms point to involvement of these organs. Of course, some activity is present in the diseased portion of the lung to be removed. This makes differentiation of the exact source of the symptoms of an active pulmonary lesion most difficult. Serial x-rays are probably the greatest help in showing which lesions of the lungs are responsible for the signs of toxicity in any given patient. However, in some instances it is impossible to decide. We believe that many so-called "spreads" of pulmonary tuberculosis following surgery, are really due to contralateral lesions or ipsolateral ones which have had undiagnosed activity or have been re-activated following resection.

It is well known that pulmonary tuberculosis is a cyclic disease, characterized by exacerbations and remissions. Care should be taken to perform the resection during a remission, is possible. It is true that exceptions to this rule exist. For example, if a tension cavity with a fluid level is present, then considerable evidence of activity of the tuberculous lesion may be found which is directly referable to the retension of infected secretions. Likewise, if there is a bronchial stenosis and the obstruction cannot be relieved by bronchoscopy, then it may be necessary to disregard the signs of infection that are present and proceed with the pulmonary resection.

B. *Treatment of ulcerative tuberculous tracheal bronchitis.* The presence of active ulcerating tuberculous tracheal bronchitis should be considered now as a contra-indication to resection and surgery postponed until the tuberculous ulceration has healed. The results of Brewer and Bogen[1] in treating ulcerative tuberculous lesions of the trachea and bronchi with intramuscular and "Aeresol" streptomycin have been most encouraging in showing prompt healing of these lesions. Although the amount of scar tissue after the streptomycin treatment seemed less than that with the conventional cauterization with silver nitrate, stenoses were not prevented. Frequently, the tuberculous ulceration of the bronchus occurs at a site where amputation and closure of the bronchial stump would be desirable at surgery. The chances of healing the bronchial stump are immeasurably improved, if the ulcerative lesion is healed before surgery is undertaken. The surgeon is no longer justified in performing pulmonary resection in a patient with active tuberculous ulceration of the bronchus, without first

giving a thorough course of streptomycin treatment. However, if only hyperemia and edema of the bronchial mucosa is present, which is either a very early stage of tuberculous bronchitis or non-specific infection, then it is safe to proceed with the resection.

If a healed bronchial stenosis is present, and there is a "back-up" of secretions beyond the stenosis, causing fever and the signs of acute infection, then it is wise to promote drainage through the obstructed portion of the bronchus by gentle dilatations of the stricture. Once the secondary infection is decreased and the fever and toxicity is ameliorated by the establishment of adequate bronchial drainage, resection may be undertaken.

C. *General measures:* Most cases of pulmonary tuberculosis have been treated with long periods of bed rest before they are considered as candidates for lobectomy and pneumonectomy. Such long periods of inactivity result in a lessened tonus of the cardiovascular system. It is desirable, therefore, to get the patient out of bed for a short time each day for a period of from two to three weeks before surgery in order that the tonus of the cardiovascular system may be restored to a near normal level. Anemia is to be corrected before the patient leaves the sanitarium, by diet and the administration of iron and liver in sufficient amounts and blood transfusions if necessary. If the blood proteins are low, high protein diet and intravenous protein preparations may be given. Wound healing is also dependent upon a normal vitamin C blood level. We are accustomed to administering from 100 mgms. to 500 mgms. of Ascorbutic acid in addition to other vitamins for the week preceding surgery.

To minimize non-specific bronchial and pulmonary infections, after operation, we routinely administer penicillin by the intramuscular and "aeresol" techniques 5 to 7 days before surgery, as well as during the postoperative period. Early reports make it seem worth while also to administer streptomycin intramuscularly so that bronchogenic spread and possible re-activation of latent pulmonary lesions may be minimized.

Operative Technique

A. *Position on the table:* The position of the patient on the operating table is of great importance, as every effort must be made to prevent the flow of purulent secretions from the lobe or lobes to be removed, to the unoperated lung. We believe that the steep Trendelenberg position, with the chest at a 45 degree angle with the horizontal, will greatly decrease the contra-lateral spread of sputum from the affected lung. The superiority of this position over the conventional prone one is readily recognized. Overholt

has used a "face down" position which, in his hands, has reduced contralateral spread from 19 to 10 per cent.

B. *Anesthesia:* The importance of a particular anesthetic agent or agents has received undue emphasis in the current medical literature. Pentothal, cyclopropane, nitrous oxide, ethylene, and ether all have had their advocates. We believe that it is the method of employment and the skill of the anesthetist in administering any given agent that is really important. Steady, deep narcosis must be maintained so that frequent bronchial and tracheal aspirations and the occasional changes in the intrapulmonary pressure can be carried out without the danger of violent cough or bronchospasm which may be followed by a bronchogenic spread. Our anesthetist employes pentothal or cyclopropane for induction and local anesthesia to aid in the rapid and smooth passage of the intratracheal tube. We have found ether the most satisfactory agent for the maintenance of long anesthesia. The apnea technique is avoided and anesthesia is maintained so that respiration is carried on partially by the patient, assisted by the anesthetist. This type of respiration, or "compensated respiration" is best for these long operations.

The flow of citrated blood into a vein is started through a large needle or cannula, when the surgeon begins the operation and does not cease until the surgical procedure is completed. The amount of transfusion administered should equal the amount of blood lost. Even though the operation has been particularly sanguinous due to the large number of vascular adhesions that were present, if the blood loss is immediately replaced, the patient stands a good chance of leaving the operating table with a systolic blood pressure above 100 and a pulse scarcely more rapid than before surgery.

D. *Surgical approach:* (1) Approach for primary pulmonary resection: The long posterolateral incision over the entire fifth rib, after the manner of Crawfoord offers an excellent exposure of the entire pleural cavity. This approach is also used for upper lobe lobectomy. For lower lobe lobectomy, a long segment of the sixth rib is removed subperiosteally from the transverse process around to the midclavicular line. In our experience these postero-lateral approaches are far superior to the anterior entrance into the pleural cavity.

2) Approach if a thoracoplasty has been performed previously: The exposure of the lung is much more difficult than when the chest wall is intact. Usually the thoracoplasty incision is re-entered and swung forward to the midclavicular line at about the level of the fifth rib. If possible, it is wise to resect the uppermost unresected rib (providing this is not lower than the seventh rib)

to gain access to the pleural cavity. The incision is then carried upward, posteriorly, paralleling the spine to gain exposure of the apex of the lung. Often what amounts to a revision thoracoplasty, with removal of a large placque of regenerated bone has to be performed before the lung is exposed. The surgeon who persists in the removal of the lung following this tedious and difficult entrance into the pleural cavity readily appreciates why it has been widely recommended to perform the lobectomy or pneumonectomy in pulmonary tuberculosis without first trying a thoracoplasty. The actual pleural space, after thoracoplasty, is smaller, thus the dissection is more difficult for there is less room for manipulation of the lung and the hilar structures are less easily exposed.

E. *Points in surgical technique:* Certain points in surgical technique will be found helpful in assuring the successful removal of a tuberculous lung and ultimate cure of the patient. Utmost gentleness in manipulation of the lung must be employed if rupture of tuberculous cavities and the expression of infected material into the bronchial tree are to be avoided. The lung must be gently separated from the chest wall and mediastinum by sharp dissection to minimize injury to the pulmonary tissue. If bleeding is encountered, hot compresses will generally control the oozing of blood; however, suture ligatures of fine silk may be necessary to control the hemorrhage from the surface of the lung or parietal pleura. From the point of view of avoidance of bronchogenic spread, the sooner the main bronchus of the lobe or lung to be removed is exposed and closed, the better. It may be advisable to place a temporary ligature around the bronchus before the final closure is accomplished. To expose the hilar vessels, one must first begin with a wide mediastinal separation. It is wise to have three sides of a main pulmonary vessel under direct vision so that digital control of bleeding is possible.

In dissecting out the bronchus, great care is taken to preserve the blood supply right down to the point of actual division of the bronchial wall. A right-angle gall bladder clamp is used to clamp the bronchus. It is applied lightly so that it does not traumatize the bronchus sufficiently to impair healing. The use of a clamp is advocated because it prevents the entrance of blood into the bronchial tree and the escape of secretions and gasses into the pleural cavity. Furthermore, in difficult cases, after bronchial section, the lung may be freed and retracted so that short pulmonary vessels are put on a stretch and ligated with safety. A second similar hemostat is placed distally to prevent contamination from the lung that is to be removed. Cutting between the two hemostats with a scalpel, the bronchus is divided. No cauter-

izing chemicals are used, as they destroy more tissue than infecting organisms. We believe that .007 tantalum wire on a half curved swedged needle of the same diameter is the suture material of choice as it produces no tissue reaction, holds the walls permanently together, and the fine swedged needle makes no larger opening in the bronchial wall than the diameter of the suture material. Three simple sutures, placed into the end of the bronchus around the clamp, are tied after removing the clamp. At intervals of approximately 1 mm. apart, other single simple sutures are placed through the two layers of the cut bronchus to draw the membraneous portion to the cartilaginous ring. It is important to turn in the cut ends of the sutures so that there is no danger of tearing an adjacent vessel from the sharp ends of the tantalum wire. An elaborate burying of the bronchial stump is not necessary. This technique is the simplest of all those mentioned in the literature. There is no other method that offers a higher percentage of successful closures.

Before closing the chest wall, the fifth intercostal nerve is resected and the fourth to the ninth intercostal nerves are crushed with a fine hemostat to prevent postoperative pain. This has proven to be a most important part of the surgical technique, for the avoidance of pain after operation allows the patient to cough freely and raise whatever sputum is present within the bronchial tree. The amount of opiates that are necessary to keep the patient comfortable are greatly decreased by this procedure and the injurious side effects from over-sedation are thus eliminated. The actual closure of the chest wall is effected in layers, using running and interrupted chromic catgut sutures.

In the case of lobectomy, both an anterior and a posterior intercostal drainage tube is employed. These tubes are connected with a closed under-water drainage system. The use of these tubes allows prompt expansion of the remaining lobe. In the case of pneumonectomies, done primarily, no drainage tubes are employed; however, if the pneumonectomy follows a thoracoplasty, the actual volume of the thoracic cavity on the operated side may be extremely small, and a temporary drainage tube is most helpful in preventing mediastinal compressions. These intercostal tubes are clamped off during the trip to the ward.

Postoperative Care

A. *Immediately, in the operating room:* Once the wound is closed and a firm dressing has been fixed in place, the patient is turned cautiously from his side to the back. This turning must be done slowly to prevent sudden mediastinal shift, which might

result in pulmonary or cardiac arrest. The success of the surgery depends upon sending the patient back to his room with the remaining lobes of the lung free of aspirated blood and secretion. The lungs are therefore carefully checked by auscultation with a stethescope and any signs of rales or retained secretions are dealt with at once. Since a catheter passed through the intratracheal tube may be left in for a longer period of time than the bronchoscope, it is preferred for the aspiration of fluids that may take some time to drain from the smaller bronchi into the main stem bronchus. Thus, with the catheter in place in the main stem bronchus, the patient may be slowly turned so that the lung with rales is uppermost and secretions from the peripheral part of the lung may have an opportunity to gravitate into the stem bronchus. We have found that the more diligent we are in aspirating even the slightest amounts of fluid material from the bronchial tree, the fewer postoperative spreads of the pulmonary tuberculosis and the fewer instances of non-specific pneumonitis will be encountered. Bronchoscopy is used if clots block a main bronchus or the catheter cannot be passed into a particular bronchus.

B. *Care on returning to the room:* Oxygen should be immediately available, to be given by the nasal catheter technique. The patient is placed in Trendelenberg position until he is awake, coughing, and the blood pressure stabilized. Carbon dioxide is administered every hour or half hour, over a period from six to eight hours to promote deep breathing, and then every three hours. It is much better to have the patient cough up his own sputum; however, if he fails in this attempt, then the surgeon should not hesitate to pass a catheter into the trachea and main bronchi by the nasal approach, after the manner of Haight.

Demerol in doses of 100 mgms. is used to control the pain. Whatever narcotic is used postoperatively, the first injection given after return from surgery should be only one-half or one-third the usual dose, to avoid depression of the respiratory center before complete recovery has taken place. If intercostal drainage tubes have been used to drain the pleural cavity, then several hours after returning to the room following surgery, 100,000 units of penicillin and 1 gram streptomycin are injected into the tube which is then clamped off for a period of six hours. Sips of water by mouth are permitted after the cessation of nausea and the patient is usually rapidly placed on a soft diet. Fluids are permitted up to 2500 cubic centimeters per day by either the intravenous or oral route. Vitamin C, one of the important factors in wound healing, is given in doses of 500 mgms. b.i.d., intramuscularly. Postoperative anemia is handled by prompt blood transfusions, so that the hemoglobin and red blood cell count are maintained

at normal levels. The intercostal drainage tubes usually cease to function in 24 to 48 hours, and so may be removed. Further accumulations of pleural fluid can be aspirated with the needle and syringe. In the case of a primary pneumonectomy, where no drainage tubes are employed, daily aspirations should be made for the first four or five days so that the mediastinum may be kept at the midline. At the time of the aspiration, penicillin and streptomycin may be introduced into the pleural cavity. Intramuscular penicillin may be stopped the second week after surgery and streptomycin in 3 to 6 weeks following the operation, unless otherwise indicated. During the early postoperative period, the patient is encouraged to move about in bed, to sit up, to move his legs, and shoulder girdle muscles so that the circulation is adequate and no muscle stiffness results and phlebostasis and phlebothrombosis are prevented.

C. *Late care:* Because we have done few lobectomies and pneumonectomies without first performing a thoracoplasty, the problem of the post-resection thoracoplasty has not often arisen in our experience. If a lobe has been removed without a primary thoracoplasty, then the chest wall should be collapsed within six weeks following the lobectomy, so that overexpansion of the remaining lobe will not result in a possible re-activation of a latent pulmonary tuberculous lesion in this lobe. In the pneumonectomies for pulmonary tuberculosis, where a thoracoplasty has not been performed previously, the mediastinum is frequently so rigidly fixed that a secondary thoracoplasty following the pneumonectomy may not be necessary; however, if there is a tendency for the mediastinum to shift toward the resected side, or for a mediastinal herniation of the remaining lung to take place, then a thoracoplasty should be promptly performed.

In general, we have insisted on six months bed rest following lobectomy or pneumonectomy for pulmonary tuberculosis. We have thought that this bed rest was necessary because pulmonary tuberculosis is usually bilateral, and that any possible activity in the remaining lobes of the lung should be thoroughly controlled before exercise was permitted. Pleural complications are probably minimized by this prolonged period of bed rest. After the patient returns to the sanitarium, he may be allowed the privilege of one visit to a bathroom or commode daily. The comfort that this affords the patient, we believe, offsets the possible disadvantage of being out of bed for this one period of time. After the sputum has been negative for a period of six months, the patient is started on a period of graded activity and exercise similar to the regimen following thoracoplasty.

Results

Surgical statistics concerning pulmonary resection, compiled from a number of clinics have little value. There are too many variable factors. Not sufficient data is published by the various authors to permit accurate classification as to the character of tuberculous lesions, the pathology of entrance of the tubercle bacilli, the manner of spread, etc. The selection of cases, the skill and method of the anesthetist, the operative technique and the postoperative care, and various other factors differ widely. Manifestly, it is of more value to report separately the results obtained in individual series than to combine them. Despite the variable factors, above mentioned, the uniformity of results is striking. In the authors' series of 26 cases of lobectomy, there are 46.2 per cent apparently well with negative sputum; 30.7 per cent alive with positive sputum and 23 per cent total deaths, of which 11.5 per cent were early postoperative deaths and 11.5 per cent were late deaths. Results following 14 cases of pneumonectomy are 41.7 per cent apparently well with negative sputum; 22.5 per cent alive with positive sputum and 35.7 per cent total deaths, of which 17.7 per cent occurred in the early postoperative period and 18.1 per cent in the late period. Our present operative technique was not used in all of these cases. As other surgeons have discovered, we anticipate a lowered mortality if the operative principles outlined in this communication are followed. Overholt[2] reports 42.7 per cent of 69 cases treated by lobectomy as apparently well with negative sputum; 18.9 per cent are alive with positive sputum, and total deaths, 14.7 per cent. Following 127 cases of pneumonectomy, there are 42.9 per cent apparently well with negative sputum; 14.6 per cent alive with positive sputum, whereas the total deaths are 28.3 per cent. In Sweet's[3] series of 27 lobectomies, 48.5 per cent are apparently well with negative sputum; 25.9 per cent are alive with positive sputum and total deaths are 25.9 per cent. His results following pneumonectomy in 36 cases are 44.4 per cent apparently well with negative sputum; 16.6 per cent alive with positive sputum and 38.8 per cent total deaths.

These figures are of value only in a general way. Time alone will give the final answer to the question of the types of cases for which resection is indicated. In our series, with few exceptions, we operated only when thoracoplasty had failed. This represents an extremely hopeless group of cases in which a cure could not be obtained by any other present day method of treatment.

SUMMARY

1) Pulmonary resection has a definite place in the surgical therapy of pulmonary tuberculosis in salvaging otherwise hope-

less cases.

2) With the exceptions cited, we prefer thoracoplasty to lobectomy and pneumonectomy in the treatment of pulmonary tuberculosis. This point of view is based on the fact that the mortality from thoracoplasty is considerably lower than pulmonary resection and to date, the late results of resection seem to offer no better chance of permanent cure.

3) When thoracoplasty fails, the following measures are considered before performing pulmonary resection:

(1) Revision thoracoplasty.
(2) Creation of extrapleural space and packing.
(3) Cavity drainage.
(4) In some cases, no further surgery.

4) Primary pulmonary resection is preferred to thoracoplasty for:

(1) Lower and middle lobe cavities.
(2) Tuberculomata and blocked cavities.
(3) Persistent bronchostenosis.
(4) Symptomatic bronchiectasis.

5) From the point of view of the pathological pulmonary changes, the type of disease most amenable to lobectomy and pneumonectomy is that characterized by fibroid and caesating lesions of the lung in which the mode of spread is principally by way of the bronchus. Since the prognosis following thoracoplasty in this type of lesion is also good, it is unusual that the surgeon is confronted with the problem of primary pulmonary resection.

6) Primarily, hematogenous or lymphogenous disseminated pulmonary tuberculosis is a contra-indication to resection, as these types of disease are progressive and removal of one part of the lung does not alter the course of the lesions in the remaining part of the lung.

7) If thoracoplasty is unsuccessful, then revision thoracoplasty, plombage, and cavity drainage should be considered before lobectomy or pneumonectomy is performed. Pulmonary resection is to be preferred to cavity drainage, if the pulmonary capacity of the patient permits.

8) In selecting any case for lobectomy or pneumonectomy, the surgeon must be certain that the patient has sufficient pulmonary reserve. Contralateral pleural thickening and diaphragmatic paralysis, along with fibrosis, calcification, and emphysema of the lung are the most important conditions in lowering the patient's breathing capacity.

9) In the surgical management of over 1,000 cases of pulmonary tuberculosis, we have performed lobectomy and pneumonectomy in 40 cases with early and late mortality of 27.5 per cent. In

approximately an equal number of cases, resection was indicated but not performed for various reasons. This represents a very hopeless group of cases where for the most part, thoracoplasty had failed, and no other treatment would cure.

10) Lobectomy and pneumonectomy are indicated in many patients where thoracoplasty has failed or obviously will not succeed. The actual number of patients upon whom it is possible to perform pulmonary resection is greatly reduced because of these factors: (1) contralateral tuberculosis; (2) instability of the contralateral lesion, and (3) low pulmonary reserve.

11) We are by no means satisfied that the indications we have presented will stand the test of time. Our point of view lies somewhere between the eager advocates for pulmonary resection, whose enthusiasm has not yet been supported by satisfactory long-time results, and those who roundly condemn lobectomy and pneumonectomy for pulmonary tuberculosis.

12) It is becoming steadily more evident that it is not technical skill alone that will lead to a higher percentage of permanent arrests in pulmonary tuberculosis. The basic tendency of the disease in each particular case is variable. Thus, the more able we become in recognizing the type of pulmonary involvement, the more accurately we will place lobectomy and pneumonectomy in their proper relationship with other therapeutic procedures.

RESUMEN

1) La resección pulmonar tiene una posición bien definida en la terapia quirúrgica de la tuberculosis pulmonar en salvar a casos que de otra manera serían incurables.

2) Con las excepciones citadas, preferimos la toracoplastia a la lobectomía y neumonectomía en el tratamiento de la tuberculosis pulmonar. Se basa este punto de vista en el hecho de que la mortalidad de la toracoplastia es considerablemente más baja que la de la resección pulmonar y en que, hasta la fecha, los resultados alejados de la resección no parecen ofrecer mejores oportunidades de curaciones permanentes.

3) Cuando fracasa la toracoplastia se consideran las siguientes medidas antes de llevarse a cabo una resección pulmonar:

(1) Toracoplastia de revisión.
(2) Creación de un espacio extrapleural y empaque.
(3) Canalización de la caverna.
(4) En algunos casos, ninguna otra operación.

4) Se prefiere la resección pulmonar primitiva a la toracoplastia en:

(1) Cavernas en el lóbulo inferior y medio.
(2) Tuberculomas y cavernas bloqueadas.

(3) Broncoestenosis persistente.

(4) Bronquiectasia sintomática.

5) Desde el punto de vista de las alteraciones patológicas del pulmón, el tipo de enfermedad más tratable por lobectomía y neumonectomía es el que está caracterizado por lesiones pulmonares fibrosas y caseosas en las que la manera principal de propagación es por las vías bronquiales. Pero desde que el pronóstico que sigue a la toracoplastia en este tipo de lesión es también bueno, es raro que el cirujano se encare con el problema de la resección pulmonar primitiva en estos casos.

6) La tuberculosis pulmonar diseminada, principalmente hematógena o linfógena, es una contraindicación a la resección, pues estos tipos de enfermedad son progresivos y la extirpación de una parte del pulmón no altera el curso de las lesiones en la parte restante del pulmón.

7) Si fracasa la toracoplastia, se deben considerar la toracoplastia de revisión, el plombaje y la canalización de la caverna antes de llevar a cabo una lobectomía o neumonectomía. Se debe preferir la resección pulmonar a la canalización de la caverna si la capacidad pulmonar del paciente así lo permite.

8) Al seleccionar cualquier caso para lobectomía o neumonectomía, el cirujano debe estar seguro de que el paciente tiene suficiente reserva pulmonar. Espesamiento pleural y parálisis diafragmática contralaterales, junto con fibrosis, calcificación y enfisema pulmonares, son las condiciones más importantes que disminuyen la capacidad respiratoria del paciente.

9) En el tratamiento quirúrgico de más de 1000 casos de tuberculosis pulmonar hemos llevado a cabo lobectomías o neumonectomías en 40 casos, con una mortalidad temprana y tardía del 27.5 por ciento. En un número de casos aproximadamente igual, se indicaba la resección pero no se llevó a cabo por varias razones. Representa éste un grupo de casos muy desesperanzados, en la mayor parte de los cuales había fracasado la toracoplastia, y que no eran curables por ningún otro tratamiento.

10) Se indican la lobectomía y la neumonectomía en muchos pacientes en los que ha fracasado la toracoplastia o es evidente que no resultará satisfactoria. El verdadero número de pacientes en los que es posible ejecutar una resección pulmonar queda muy reducido por los factores siguientes: (1) tuberculosis contralateral; (2) inestabilidad de la lesión contralateral y (3) baja reserva pulmonar.

11) No estamos seguros de que las indicaciones que hemos presentado resistirán la prueba del tiempo. Nuestro punto de vista no coincide ni con los que abogan vehementemente por la resección pulmonar, cuyo entusiasmo no ha sido apoyado todavía por

resultados satisfactorios por suficiente tiempo, ni con aquellos que francamente condenan la lobectomía y neumonectomía en el tratamiento de la tuberculosis pulmonar.

12) Se evidencia cada vez más que no es la destreza técnica sola lo que producirá un porcentaje más alto de detenciones permanentes de la tuberculosis pulmonar. La tendencia fundamental de la enfermedad varía en cada paciente. De manera, pues, que mientras más desarrollemos la habilidad de reconocer el tipo de lesión pulmonar, con más exactitud podremos colocar la lobectomía y neumonectomía en su propia relación con otros procedimientos terapéuticos.

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Discussion

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The improvement in the technical procedures of lobectomy and pneumonectomy has resulted in a steady decline in the mortality rate. The use of antibiotics, whole blood replacement and measures to prevent a bronchogenic spread of the disease are as important as the surgical procedure. The ability to perform a successful lobectomy or pneumonectomy does not of itself constitute an indication for such surgical procedures.

Pulmonary tuberculosis is usually not a localized disease, therefore extirpative surgery or local removal, except in selected cases cannot be expected to produce a cure. When considered as a method of correcting or overcoming the complications of tuberculosis, extirpative surgery would seem to have a wider application.

It has been stated by Dr. Brewer that resection is not a competitive form of treatment, but should be used when the standard collapse methods have failed or obviously will fail. I believe that most of us are in general agreement with this statement and it is only in those "anticipated failures" where the absolute indications are lacking that any disagreement occurs.

The indications for resection are not absolute although they are

becoming clearer. We now limit our resections to those patients falling into three groups: (1) Tuberculoma, (2) Thoracoplasty failures and (3) Anticipated failures of which the chief ones are: (a) inaccessable cavities and (b) marked bronchial stenosis.

In our small series of 20 cases only 40 per cent of the patients are alive and well, three years after resection. This does not compare favorably with our thoracoplasty statistics. I believe the continued observation of these patients will lead to more definate indications for resections.

I want to congratulate Dr. Brewer upon his excellent paper.

Benign Pulmonary Histoplasmosis*

A Case Report With a Brief Review of the Literature

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Until recent years histoplasmosis has been considered a fairly rare and always fatal disease; however, evidence is steadily accumulating that a benign phase of the disease exists.

Darling was the first to describe the disease and reported three cases (1906,[1] 1908,[2] 1909[3]). He suggested that the organism responsible for the pseudotuberculous lesions in the lungs be called *Histoplasma capsulatum* and that the disease be called histoplasmosis. DeMonbreun,[4] in studying the first case of histoplasmosis reported from Tennessee by Dodd and Tompkins,[5] was the first to describe the cultural characteristics of the organism and, by satisfying Koch's postulate, proved that the fungus is responsible for the disease histoplasmosis.

There are several excellent reviews of the literature on histoplasmosis; therefore, we will mention only those articles that seem pertinent to this report. Those further interested may refer to the excellent reviews of Meleney,[6] Iams, Tenen and Flanagan,[7] Parsons and Zarafonetis,[8] and Moore and Jorstad.[9]

Cases have been reported from Europe, the Southwest Pacific, South America, Central America and nearly every section of the United States. The greatest number of reported cases has been from the mid-western area of this country.

The last known survey of the literature on histoplasmosis is by Ziegler[10] who reported a case from Pennsylvania in June of 1946. His case brought the number of reported cases to 79. Since then we know of at least 19 additional cases being published,§ including the one reported here. These additional cases are tabulated (See Table 1).

The reported cases have nearly all been generalized or miliary in type. Those cases that were not fatal have shown localized

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§No doubt there are other reported cases that have not come to our attention.

lesions usually characterized by ulceration of skin or mucous membranes. As yet little is known of the pathogenesis of this disease. However, the probability is that it will parallel tuberculosis as closely as it does clinically.

The symptomatology varies according to the organs that are involved. Since the pulmonary disease closely resembles tuberculosis of the lungs, it is natural that the symptoms of pulmonary histoplasmosis are similar. Cough, purulent sputum, chest pain, fever and night sweats have been reported as symptoms.

The published work of Palmer[22,24] and of Christie and Peterson[23] shows that the area with a high incidence of tuberculin negative, histoplasmin positive persons with pulmonary calcifications corresponds fairly well with the area in the United States from which most of the cases of histoplasmosis have been reported.

Parsons and Zarafonetis[8] have shown that the disease is more frequent in males than in females, about 3½ to 1 in all ages, and about 7 to 1 in persons over 10 years of age. While they have

TABLE I

Cases Reported Since Ziegler's Review in June, 1946

Case	Reported by	From	Date Published	
80	Davis & Neff - 12	Missouri	Feb.	1946
81	Davis & Neff - 12	Missouri	Feb.	1946
82	McLeod, Emmons, Ross & Burk - 13	Virginia	March	1946
83	McLeod, Emmons, Ross & Burk - 13	Virginia	March	1946
84	Madureira - 14	Brazil	May	1946
85	Madureira - 14	Brazil	May	1946
86	Madureira - 14	Brazil	May	1946
87	Madureira - 14	Brazil	May	1946
88	Kuzma & Schuster - 15	Wisconsin	June	1946
89	Swan & Finnegan - 16	Wisconsin	Aug.	1946
90	Seabury & Drygas - 17	Michigan	Aug.	1946
91	Seabury & Drygas - 17	Michigan	Aug.	1946
92	Iams, Keith & Weed - 7	Minnesota	Dec.	1946
93	Mider, Smith & Bray - 11	Virginia	Jan.	1947
94	Conklin & Hankins - 18	Nebraska	March	1947
95	Curtis & Cawley - 20	Michigan	April	1947
96	Thomas & Mitchell - 19	Illinois	May	1947
97	Lam & Price - 21	Honolulu	May-June	1947
98	Johnson & Batson	Alabama	?	1947

shown that every organ has been involved in one case or another, those most frequently found involved at autopsy are in the order of their frequency, the "spleen, liver, visceral lymph nodes, lungs, bone marrow, oral mucosa, adrenals, gastrointestinal tract, peripheral lymph nodes, kidneys and larynx." Vegetative endocarditis was found in three patients. They also have shown that "the most common signs and symptoms in the order of their decreasing frequency are: fever, hypochromic anemia, hepatomegaly, splenomegaly and lymphadenopathy."

In view of these facts, it would seem that one should consider histoplasmosis in any case in which there are various combinations of fever, nodular or ulcerative lesions of the skin or mucous membranes, generalized lymphadenopathy, hepatosplenomegaly, anemia and leukopenia, and low blood pressure. With pulmonary disease resembling tuberculosis but with negative sputum and negative tuberculin tests one certainly must include histoplasmosis in the differential diagnoses. This is particularly true in the geographical areas with a high incidence of pulmonary calcifications in persons with negative tuberculin tests and positive histoplasmin tests. It goes without saying that one cannot diagnose a disease without first thinking of it.

Skin testing with histoplasmin and old tuberculin should be done on all patients suspected of having histoplasmosis. When possible, one should also skin test the patient with coccidioidin and blastomycin in order to rule out possible cross immunologic responses. A positive histoplasmin test should not be interpreted as being diagnostic but is highly suggestive of antecedent infection with *H. capsulatum* or another immunologically related organism.

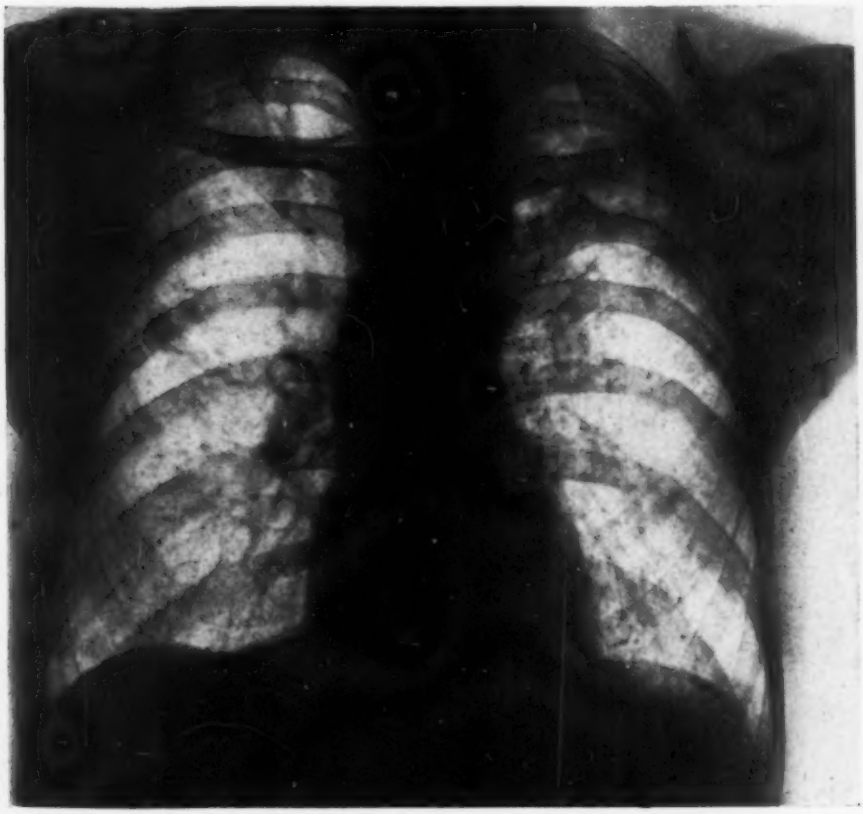
The diagnosis may be confirmed by culture or by microscopic demonstration of the organism in tissue preparations. The yeast cell phase of the fungus may at times be seen by microscopic examination of bone marrow or peripheral blood that has been stained with Wright's or Giemsa's stain. These yeast cells appear singly or in groups as small oval bodies surrounded by a capsule and located within the cytoplasm of large phagocytic cells. It is also possible to demonstrate these organisms in smears of material from ulcerative lesions, biopsied lymph nodes, or visceral punctures. *H. capsulatum* may be cultured from any of the above material by inoculation onto blood agar and Sabouraud's medium. Cultures should be allowed to grow in duplicate at room temperature and at 37° C. At incubator temperature yeast cell colonies may appear, whereas at room temperature the organism always grows in the mycelial phase. The latter culture should not be discarded until the appearance of tuberculate chlamydospores, which establishes the mycologic diagnosis. It is important that

all cultures be sealed to prevent drying and that cultures not be discarded as negative until a period of 6 weeks has elapsed.

When culturing sputum one should mix penicillin and streptomycin with the material to a final concentration of 1000 units of each antibiotic per cc. The mixture should incubate at 37° C. for 1 hour after which time it is inoculated onto culture media. The addition of penicillin and streptomycin suppresses the growth of common bacterial contaminants.

The growing number of reported cases of histoplasmosis and the close resemblance of its pulmonary type to tuberculosis makes the case we are about to present most important. Clinically, this case had all the earmarks of pulmonary tuberculosis but, as will be shown, such a diagnosis could not be established.

M.J.C., a 64 year old farmer residing in North Alabama, was admitted to Vanderbilt Hospital on July 16, 1947. He stated that, except for pain in his left sacroiliac region, and a chronic nonproductive morning cough which had begun following pneumonia and typhoid fever at age 11, he was well until 10 months prior to admission. At this time his cough became more severe and was productive of small amounts of purulent sputum. He had chest pain which he was unable to describe but which



covered an area extending from the apex of the left axilla to the left base between the anterior and posterior axillary lines. This pain was not influenced by exercise or breathing. There was also an associated pain in the region of the left elbow.

The sputum was greenish yellow, odorless and tasteless, and at no time contained blood. The 24 hour output was 2 or 3 tablespoonfuls. During 6 months preceding admission he developed paroxysms of coughing both day and night. He had no night sweats. He was easily fatigued and lost 7 pounds despite a good appetite. There is no history of tuberculosis in his family and no history of contact with that disease. His family and personal history are otherwise unimportant. His previous health and illness review is not remarkable. His system review reveals traumatic blindness in the right eye since age 20; a history of carious teeth; a right inguinal hernia; and no veneral disease.

Physical examination revealed a well developed and nourished white man apparently of stated age. His skin was dry and slightly scaly and without ulcers or eruptions. He had a corneal opacity in the right eye with which he could only distinguish light. His oral hygiene was poor. The teeth were markedly carious and there was moderate gingivitis without ulceration. His thorax showed slight asymetry with flattening on the left where there was some lagging with inspiration. The percussion note was impaired slightly over the left upper chest where the breath sounds were suppressed and bronchovesicular. There were no signs of cavitation and there were no rales. Otherwise examination revealed only a moderately enlarged, smooth prostate without tenderness and slight clubbing of the fingers.

The remainder of the examination was not pertinent to this report. His temperature, pulse and respiration curves were normal during the 16 days hospitalization.

The x-ray was reported as follows: "There is a cavity in the left apex overlying the anterior tip of the first rib and in the first anterior interspace which measures about 3 x 4 cm. with considerable pleural thickening along the chest wall in the apex extending downward along the lateral chest wall. Each costophrenic angle shows some blunting. There are fibrotic infiltrations in the right apex and in the right lung field which extend across the right mid-lung field into the periphery. Scattered through each lung field are multiple small rounded calcified lesions. Heart is within normal limits as to size and shape. Impression: Pulmonary tuberculosis with cavitation in the left apex."

Laboratory Data: Urine cloudy. Specific gravity 1.017. Reaction acid; no albumin; no sugar; microscopic examination showed an occasional W.B.C. but no R.B.C. or casts. Blood: R.B.C. 4,350,000; W.B.C. 6,400; hgb. 12 gm.; differential normal; Kahn negative. N.P.N. 33; sugar 84; total serum protein 7.34, serum albumin 4.41, serum globulin 2.93. Stools showed no blood, ova or parasites. Sputa on three direct smears were negative for tubercle bacilli. Gastric washings negative for acid-fast bacilli. Guinea pig inoculation with gastric washings negative for tubercle bacilli. Blood culture negative.

O.T. Skin test (Montoux) 0.1 mg. and 1.0 mg. negative. Coccidioidin 1.0 mg. negative. Histoplasmin 0.1 cc. of 1:100 dilution 4+ (approximately 12½ cm. in diameter).

Because of the positive histoplasmin test, the negative sputum and the negative tuberculin tests histoplasmosis was suspected. The patient's

sputum was cultured by the technique described above.* Many colonies of *H. sapsulatum* appeared in approximately 10 days and tuberculate chlamydospores were identified after 6 weeks.

No treatment was administered to this patient other than by symptomatic and supportive measures including bed rest and a high caloric, high vitamin diet.

He was discharged from the hospital after marked reduction of cough but returned in 3 months for reevaluation of his condition. This examination was made November 1. He had regained 7 pounds but his productive cough was essentially unchanged. Physical examination was the same as previously and the x-ray of his chest was unchanged.

We did not treat the patient with any of the antimony or diamidine preparations because they have not yet proved of value and because he was responding favorably to the bed rest and general supportive measures.

This is a case of pulmonary histoplasmosis without demonstrable evidence of involvement of other organs. It is further evidence that a benign type of histoplasmosis exists, adding to the mounting evidence compiled by Christie and Peterson.[25a,b,c,d] Exception may be taken to calling this a benign case but whether benign or mild it appears to be a link between the cases showing calcifications with positive histoplasmin and negative tuberculin tests, and those showing overwhelming generalized infection.

We are privileged to mention two cases reported by Christie and Peterson that point toward benign phases of this disease, primarily considered universally fatal.

The first[25a] is that of a well developed and nourished 10 month old infant with bilateral subdural hematomata who died following an attempt at excision of the membrane. At autopsy a small calcified nodule 3 or 4 mm. in diameter was found in the lower lobe of each lung. The hilus glands were enlarged but showed no caseation. Microscopic sections were made of the calcified areas and the enlarged hilus lymph glands in both lungs and no tubercle bacilli were found; however, they did show tubercle-like lesions which contained large mononuclear cells. The fungus was cultured from the calcified lesions.

Another case[25a] was that of an 8 month old infant with hydrocephalus, meningocele, and spina-bifida. There was no history of contact with tuberculosis. At autopsy sections from the lung through a tubercle in the periphery of an area of interstitial pneumonitis revealed large mononuclear cells in which the yeast cells of *H. capsulatum* were found.

There have been other cases[25a] in which the principal disease was unrelated to histoplasmosis and in which equally positive evidence of benign histoplasmosis was found. Seventeen cases of histoplasmosis have been diagnosed at the Vanderbilt University Hospital and 6, including the one here reported, have been recorded in the literature. This non-disseminated phase of the

*The isolation and identification of the organism was carried out by Dr. David McVickar.

disease no doubt exists in other endemic areas; therefore, it would seem likely that other cases have been recognized but not reported, also there are probably many that have not been suspected or recognized.

SUMMARY

1) A brief summary of the pertinent literature on histoplasmosis has been presented.

2) The symptomatology and diagnostic procedures have been discussed.

3) Nineteen recently reported cases of histoplasmosis have been tabulated.

4) A case of benign or mild pulmonary histoplasmosis has been presented.

RESUMEN

1) Se ha presentado un breve resumen de la literatura pertinente a la histoplasmosis.

2) Se han discutido la sintomatología y los procedimientos para el diagnóstico.

3) Se han arreglado en forma de tabla diecinueve casos de histoplasmosis sobre los cuales se ha informado recientemente.

4) Se ha presentado un caso de histoplasmosis pulmonar leve o benigna.

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Pitfalls in Dealing with Cancer Statistics, Especially as Related to Cancer of the Lung*

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It is a great honor to be asked to address such a distinguished body of physicians, and I am grateful to your program chairman for this invitation, because it not only gives me an opportunity of sharing with you some of my ideas about collecting and analysing cancer data, but also because it has furnished me a vacation here with my husband, and both of us are very grateful to Dr. Banyai for this opportunity.

Although my title might lead you to think that my address will be mathematical, that is not the case. I have not the training in statistics to deal with the subject in a highly complex manner; I shall point out what are common fallacies in our treatment of cancer data. I should say that 90 per cent of statistical analysis is the application of common sense. The greatest source of error in both the collection and analysis of the data is a lack of proper controls. The point at which this is first noted is in the information inquired for in the clinical history. We attempt to elicit facts which we consider as significant in the past history of the patient, and the significance relates entirely to what we think are causative factors in producing the cancer. Inasmuch as we do not know the cause of most cancers, it is obvious that what we ask for is what we think is contributory. It is small wonder then that we find our ideas about contributing factors confirmed. We are not likely to correct any mistakes in our ideas, nor are we apt to add new ideas.

As an example of this type of error, we might look at the cancer forms put out by the American College of Surgeons for cancer in various parts of the body. The patient with rectal cancer is asked about constipation, hemorrhoids, etc., the patient with skin cancer is asked about his exposure to the weather; the patient with breast cancer is asked about abnormalities in lactation history, and the patient with cervical cancer is queried as to the number of child-

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ren, and miscarriages that she has had. But the woman with breast cancer is not asked about her constipation, and the man with cancer of the prostate is not asked about exposure to the weather. The result is that we find that a given percentage of rectal cancer patients have a history of constipation; but we do not know whether that is higher, lower or the same as the percentage of prostatic cancer patients who are constipated. If we find a history of breast infections in a certain percentage of women with breast cancer we do not know whether that is greater, less or identical with the percentage of breast infections in women who have cervical or gastric cancer. Our methods of collecting data on the incidence of conditions which may be thought to be productive of any given type of cancer tend to confirm us in the ideas we already had but they do not lead us to make new discoveries, and they do not offer us a control as to the significance of the information we collect.

In order to have our data and the deductions made from them of value, we must know more than the incidence of any factor in the history of our particular group of patients; we must know the incidence of the same factor in patients not suffering from the disease we are studying. We may find that 95 per cent or 99 per cent of our lung cancer patients smoke; but what does that mean if we find that 95 or 99 per cent of all men smoke? Ninety per cent of women with cervical cancer are said to have had children. What percentage of women without cervical cancer have had children? I once heard a man make the statement that he thought that some chemical in tobacco was productive of cervical cancer, because every woman who had come to him in his practice with cervical cancer worked in a cigar factory at some time in her life. On inquiry, I found that all the women in the town in which he practiced worked in a cigar factory at some time in their lives, so that it could equally well be claimed that rolling tobacco leaves was the cause of fractures, babies, indigestion and corns in the women of that community.

One sees papers published to the effect that in a given district lung cancer is oftenest found in the workers in the pottery factories; in other districts, lung cancer will be said to occur almost exclusively in the coal miners, etc. Accompanying this should be a statement as to what percentage of the male workers in the first community were from the pottery plants; in the second, what percentage of the male workers in the district were coal miners. As long as we do not control our data, by asking the same questions of those not suffering from the disease as are asked of the patients with the disease, we cannot legitimately assume that the positive history of a trait has any significance. I ask of all the cancer

patients I interview, no matter where their cancer is located, the same questions, omitting only the menstrual and obstetrical data when interviewing male patients. I inquired of a man with prostatic cancer whether he was unduly exposed to the weather. He said that he was an engineer on a freight train, and that for thirty years he had been exposed to wind and sleet, rain and sun, with his head and shoulders out of the cab window. But he had no skin cancer. Had I elicited that history from a man with cancer of the skin of the face, it would have at once been assumed that his exposure had produced the lesion. I inquired further of him whether he was constipated. His answer was "You can guess that for yourself. When you are in a cab of an engine, you just learn to be constipated." But he did not have rectal cancer. How often would we elicit a positive history of a condition thought to be productive of a certain type of cancer in patients with other types of cancer, or with none at all, if we took the trouble to ask for it? We ask for what we want to find, and then reason that because we have found it, it is of significance.

We make the same type of mistake in collecting information on family history of cancer. One of the moot questions in connection with cancer is whether there is a genetic basis for cancer in general or for specific types of cancer. Again the forms referred to ask for "Family history of cancer." There are two possible answers, positive and negative. If positive, the inquirer probably asks what relatives had cancer, he may even go so far as to ask what relatives had what type of cancer; but that is putting a stretch upon the imagination. But if the answer is negative, it is left there; nothing is done about finding out why it is negative. It may be negative for one of several reasons. (1) It may be negative because no close relative who is dead was known to have had cancer when he died, and no close relative living has a known cancer. (2) It might be negative because the patient was uninformed. Since the policy of many of the profession is to conceal the fact that the patient has cancer, a positive history may be obscured by the patient's ignorance of the facts. Just recently, I had occasion to correspond with two sisters of the man with prostatic cancer just referred to. Both informed me that there was no cancer in their family, yet of the nine relatives whose names I could obtain in that family three had proved diagnoses of cancer. (3) The family history may be said to be negative because there was no family to show the disease. Thus the man may have prostatic cancer, yet all his brothers may have been sisters, and all his uncles may have been aunts. If the possible genetic factor could be inherited through the female, he might have received it from his mother who could not have prostatic cancer. He might have received it from his

father, who died at 35 before the age at which he would develop the cancer. (4) The family history may be said to be negative, because the patient interviewed may be the first in the family to have developed it. Had the history been reinvestigated 15 or 20 years later, several members of the family may have become affected. When a man states that he has a negative family history for gastric cancer, but that he has 8 brothers and sisters living, all free, and had ten uncles and aunts on both sides of the family, all free, it may be a very different history from that of another man who gives the same history exactly. In the first case, the man may have been the youngest of the family, and his mother and father were the youngest members of their respective families. All his relatives lived to an age older than that at which he developed his gastric cancer. The second man may have been the oldest of his family, and his father and mother the oldest of theirs, and all the aunts and uncles may have died before the age at which he showed his cancer, and his own brothers and sisters have not yet reached it. Therefore, studies purporting to give an idea as to the role which heredity plays in cancer, and which state that 35 per cent let us say, of patients give a positive family history, while 65 per cent give a negative family history, or vice versa, are very likely to be worthless.

Such studies, to be of any value, should state how many mothers of cancer and control patients of the same age, lived to be in age group 30 - 34; 35 - 39, etc., and how many mothers in each age group in the cancerous and non-cancerous divisions had cancer; how many fathers, uncles, aunts, first cousins, brothers and sisters, etc., lived to be in each age group and how many of them developed cancer and at what ages. Thus the woman with breast or uterine cancer, with no female relatives except a mother who died young, or the man with prostatic cancer with no male relatives except a father, would have that family properly weighted in the study, and the statement that the family history was negative would be given no more importance than was due it. When we consider that about 45 per cent of cancers in women and about 11.5 per cent of cancers in men are in the reproductive organs or breast and hence incapable of occurring or very unlikely to occur in the opposite sex, we can see how often a family history can be negative, because there was no relative of the appropriate sex and age to show the cancer. This reminds me of a professor who said to me on one occasion after I had been lecturing on the influence of heredity in causing cancer, "My great-grandmother, my grandmother, and my own mother all had cancer of the uterus. If what you say about heredity is true that is a very black outlook for me." I assured him that I could promise him immunity to that type of

cancer. He was in the position of one being all dressed up and no place to go.

Another factor which one is likely to forget in analysing cancer data for possible genetic factors, is that it is quite possible to find more cancer among relatives of non-cancerous persons than among the relatives of persons with cancer. This is because the non-cancerous persons have as their cancerous relatives all of the persons with cancer, whereas the cancer patients have the cancer relatives minus themselves. Let me illustrate. Consider a litter of 11 mice from a strain which shows lung cancer. Three of the 11 mice have cancer, 8 are free. Each mouse in the litter has 10 sibs, that is brothers and sisters. Each non-cancer mouse has 10 sibs, 3 of which have cancer, an incidence of 30 per cent. Each cancer mouse, on the other hand, has 10 sibs, only 2 of which have cancer, an incidence of 20 per cent. Therefore, non-cancerous mice in this litter have 1½ times as many cancer relatives as have the cancer mice. Of course, that illustration was not exactly comparable to what we do when we collect data on human families, because we do not deliberately pick as our non-cancer control, a sibling of a cancer patient. But cancer as a group of diseases is so common that we can scarcely pick a control case who did not have a relative with cancer. Only when we study the genetic factors in a particular type of cancer, which is not so common that we are almost guaranteed to pick as a control a person from a cancer family, will we be able to use this method. I wish to point out, however, that with a cancer that is very common, or if one is studying genetic factors of cancer in general, one can easily find that non-cancerous controls have a greater incidence of cancer among their relatives than have persons with cancer themselves.

Another type of improper control for analysing cancer data arises through ignoring the fact that every cancer has a specific age incidence, and sex predilection. I have already mentioned breast, uterine and prostatic cancers, but other types of cancer, not of the generative organs, have marked sex predilection. Cancer of the lung is a good example. It occurs four times as frequently in the male as in the female. If we desire to make any study of causative factors in lung cancer we must be sure that our control group is comparable to our experimental group. Again I will take an example from the literature. A worker was investigating the possible role of silicosis in inducing lung cancer. He compared the incidence of lung cancer in a group of 50 cases of silicosis, and in a large necropsy group of 4500 "unselected" cases from a general hospital. He found that lung cancer was 7 times as frequent in the silicosis group as in the unselected necropsies. This is an

excellent example of misunderstanding as to what is meant by "random" sample. Because the 4500 necropsies were "unselected" the worker thought that he had a good control group. As a matter of fact, in order to have a good control, he needed to select very carefully from these 4500 necropsies, those which he was to use as his standard. He forgot two things: (1) that lung cancer is 4 times as common in the male as in the female and that all his silicosis cases were males, therefore his unselected necropsies should have been highly selected to contain only males. Assuming that half of his 4500 necropsies were females, and that among them one fifth of the lung cancers occurred, one can easily show that had his control group been all males as was his silicosis group, lung cancer would have been only 4.8 times as common among the silicosis patients as among the general necropsy group instead of 7 times as he found it. (2) The second thing he forgot is that silicosis does not develop until 15 or 20 years of exposure have passed by. That placed all his silicosis patients in the late forties or early fifties, just when lung cancer becomes most common. Many of his general necropsy group were in the age range below 45, hence not in the lung cancer age. He should have selected only those males from the necropsy group who matched the age distribution of his silicosis patients. If he then found a significantly higher percentage of lung cancer among his silicosis patients he could have suggested a relationship between the two. Until that control group is properly studied, his results are valueless.

In the field of treatment we find many studies with figures not properly controlled. For example, Dr. A. may state that with his treatment of cancer of the prostate, he has 50 per cent 5 year survivals, 10 per cent 10 year survivals, etc. Dr. B. on the other hand with a second treatment has only 35 per cent 5 year survivals, and only 3 per cent 10 year survivals. (These figures are chosen for the sake of illustration and any resemblance between them and published data on prostatic cancer is purely coincidental). We would be led to conclude that Dr. A.'s treatment was superior. What we should be told, however, in each instance is how many of their patients would have survived 5 or 10 years even had they had no prostatic cancer. If Dr. B.'s patients are for the most part nearing 80 years of age, while Dr. A.'s were in the neighborhood of 55 or 60, we understand that the first treatment may not have been better, Dr. A. was merely more fortunate in picking them young.

In the treatment of lung cancer, one surgeon may have much better results than a second man has, but we need not conclude that the former is necessarily the better surgeon. He may merely have had better patients, with less disease in the other parts of

their body. A group of patients who die of cerebral hemorrhage 6 months or a year after their lung has been removed for cancer, can spoil a good surgeon's survival rate. Here again carefully controlled data are necessary before we can arrive at accurate conclusions.

Nowhere, however, has there been more muddled thinking than on the question of whether lung cancer has increased or not. The question has been impossible to answer because we have no standard of comparison. We have no idea of how many lung cancers were undiagnosed 25 or 15 years ago, and no idea as to how many are undiagnosed today. If 50 per cent were not recognized then and only 25 per cent were not recognized now, we could feel that lung cancer had increased by 50 per cent over what it had been in the past, whereas it had not increased at all, we merely recognized it when we saw it. There is no doubt that it is being diagnosed more frequently, and that the apparent increase is even greater than can be accounted for by increase in age of the population.

Various methods are resorted to in order to show that lung cancer has increased, even beyond the improvement in diagnosis and increase in age. The percentage of lung cancers in hospital admissions is said to have increased. But the opening of a new pediatrics wing or maternity ward can lower the percentage of cancers in hospital admissions, and the founding of a new surgical unit can increase it. The percentage of lung cancers among necropsies has been thought to be a sounder method of proving the increase. But that is no more valuable than the first method. The zeal with which the physician presses for a necropsy is of prime importance in determining which cases come to necropsy; and the zeal of the physician is directly proportional to the lack of clarity in the diagnosis. Hence the clear cut case of pneumonia, tuberculosis, or infection following trauma may not come to necropsy; but the obscure case, among which will be some lung cancer patients, will be necropsied. A third method supposed to be superior to the other two, is the estimation of increase of lung cancer cases among the cancer necropsy cases. Breast, uterine, rectal or prostatic cancer can be diagnosed by biopsy or at operation. If the patient dies, the disease has already been proved pathologically, and there is no urge to have a necropsy. The patient with vague symptoms, perhaps with undifferentiated cancer in a lymph node, will come to necropsy, and may prove to be a lung cancer. Thus since not all cancer cases were necropsied then or now, such increase may be spurious. Although as chest men you are no doubt seeing far more cases of lung cancer, there is not a single criterion by which we can prove that lung cancer is actually affecting more persons in the lung cancer age groups now than

it did 25 years ago. You may merely be seeing more of it, because you recognize more of it, and not because there is more of it to recognize.

Almost any conclusion which one wishes to draw about cancer can be supported by figures, if incorrectly collected or interpreted. Without any elaborate statistical procedures of analysis, and with only a large amount of sense of what constitutes a good control, one can avoid many of the more obvious pitfalls which beset the person working with cancer data. The two things to be remembered are, (1) that in order to have a good control group for study in comparing with a cancer group, the two groups should be as near alike in all respects save the cancer as it is possible to have them. The second thing is that partial truths may be as misleading as falsehoods. The best thing to do is to consult a competent statistician in the collection of your data, and in planning the analysis later. The old saying that "Figures can't lie, although liars can figure" is not true. Figures can lie, if by lying, we mean the creation of a false impression. It is incumbent upon us, that we recognize a well controlled conclusion about cancer when we see it in print, so that we may not be misled into following false gods, and that we know how to arrive at a properly controlled conclusion that we may not place a stumbling block in the path of others who are following after us and trusting in the truth of our conclusions.

SUMMARY

The prime requisite of data assembled to elucidate any problem is that it be collected without prejudice. This requires that the same information be gathered from the group for which it is supposed that the data are pertinent, and from the control group. If exposure to sunlight is regarded as a potential source of skin cancer, the presence of such exposure must be inquired into not only in skin cancer cases, but in patients not having skin cancer. Failure to observe this rule will naturally produce biassed data. Forms in use by medical organizations for collecting information on possible causes of cancer of any specific organ ignore to a large extent this primary rule, asking for the presence of etiological agents where they are expected and failing to ask for the same agents in control groups, furnished by patients with cancers in organs other than the one supposed to be caused by the agent. This procedure confirms preconceived ideas, and militates against advancing new ones.

The second point to be noted is that the control group should correspond as nearly as possible in all respects with the group under investigation, with the single exception of the etiologic fac-

tor being investigated. If silicosis is being considered as a causative agent in lung cancer, the control group should be as nearly like the experimental or observed group as possible in sex, age distribution, race, facilities for diagnosis, other possible carcinogenic factors, etc. The only point in which the control group should differ in an ideal study would be that they were not exposed to free silica, whereas the experimental group was. The incidence of lung cancer could then be compared in the two groups of patients. This necessity is often ignored; and a "random" control group is obtained for comparison on the assumption that any group taken at random is a good group for comparison. Fallacious results based on such studies are discussed briefly.

RESUMEN

El principal requisito de datos coleccionados para dilucidar cualquier problema es que sean copilados sin prejuicio. Esto requiere que se recojan los mismos informes del grupo al que se supone que tocan los datos, como del grupo de testigos. Si se considera que la exposición al sol es una causa potencial del cáncer de la piel, se debe investigar la presencia de tal exposición no sólo en los casos de cáncer de la piel sino en pacientes que no sufren de cáncer de la piel. La falta de observar esta regla naturalmente producirá datos parciales. Los esqueletos usados por organizaciones médicas para copilar informes acerca de las posibles causas del cáncer de un órgano específico no hacen mucho caso de esta regla primordial, pues investigan la presencia de agentes etiológicos cuando se los espera, pero no preguntan por los mismos agentes en grupos de testigos proporcionados por pacientes con cánceres de órganos diferentes del que se supone ser causado por el agente en cuestión. Este procedimiento sólo confirma ideas concebidas de antemano y se opone al desarrollo de nuevas ideas.

El segundo punto que se debe notar es que el grupo de testigos corresponda lo más posible en todo respecto con el grupo bajo investigación, con la única excepción del factor etiológico que se está investigando. Si se está estudiando la silicosis como un agente causal del cáncer del pulmón, el grupo de testigos debe ser lo más semejante al grupo experimental u observado en cuanto al sexo, edad, raza, facilidades para el diagnóstico, otros posibles factores carcinógenos, etc. En un estudio ideal, el único punto en que el grupo de testigos debería diferir sería en que ellos no estuvieron expuestos a la sílice libre, mientras que el grupo experimental sí lo estuvo. Podría entonces compararse la frecuencia del cáncer del pulmón en los dos grupos de pacientes.

Respiratory Volume Changes in the Pulmonary Blood Vessels in Relation to Artificial Relaxation Therapy*

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I. *The Bridge*—the lesser circulation.

It is helpful to think of the pulmonary vascular system as a one-way bridge for blood between the right and left hearts. The unique feature of this blood bridge is its motile environment. No other subdivision of the vascular system is surrounded, as are most of the parts of the pulmonary arterial and venous trees, by a web of passively expanding and actively contracting air-tissue which perpetually and usefully increases and decreases their capacity with each respiratory cycle. During inspiration this web, by pulling upon the walls of these blood vessels, to which it is directly attached, tends to lengthen and widen them, so enlarging their content and diminishing the resistance to the flowing blood within them, and hence lowering the pulmonary blood pressure; and all this it does by energy generated in the musculature of the chest wall and diaphragm, and transferred by alveolar air pressure. During expiration, on the other hand, through the relaxation of this enveloping tissue and the coincidental contraction of their own intrinsic elastic fibers, these vascular parts are shortened and narrowed, their content is diminished and the pulmonary blood pressure raised. The continued alternation of these lung-motivated processes of capacity increase and decrease, in which the pulmonary capillaries share passively, confers a pumping action upon the pulmonary vasculature which is most marked in the vigorously exercising young athlete. Viewed in this way the bridge of the normally functioning individual is propulsive, acting as an accessory heart. Let us examine first the evidence favoring inspiratory enlargement of these channels.

(A) *Inspiratory lengthening and widening.*

That the pulmonary arteries and veins must elongate during inflation is clear from merely observing a fresh normal mammalian lung as it is being blown up, for its major dimensions as well as

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those of individual lobes increase. The vessels could no more remain unstretched in this process than could the bronchi (Macklin 1932a, 1932b, 1936). It is clear, too, that they cannot become narrow during this extension, as do rubber tubes when stretched, for there is no inspiratory impediment to the flowing blood such as would then certainly ensue. Yet we should not dismiss the matter as taken for granted like gravitation, for an analytical consideration gives us much insight into the way in which the bridge is made more capacious in inspiration. Fortunately when entering air is swelling the air-tubes, entering blood is synchronously swelling the blood-tubes. The act of breathing brings blood as well as air into the lungs.

Histology: Examination of microsections of the inflated lung shows that all but the hilar trunks and the finest tips of the branches of the pulmonary arteries and veins are so attached to their enveloping air spaces that the inflation of these must not only lengthen the vessels but widen them, or at least prevent their being narrowed. As an example of this intimacy of relation of vessel-wall and lung stroma, figure 1 is presented. It is a photomicrograph from a section cut from the inflated lung of a rabbit and shows small veins v, v_1 and v_2 to which are attached the surrounding air sacs, *as*. The walls of the blood vessels are enwrapped by the approximated bases of the alveoli to which they are firmly bound. It is clear that the row of air sacs enveloping v_1 for instance could not expand without increasing the length of v_1.

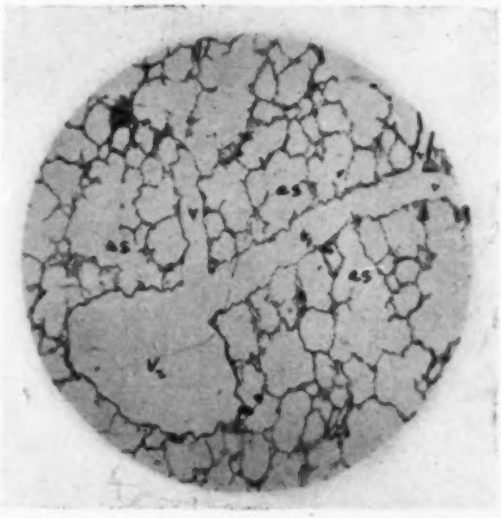

FIGURE 1: Pulmonary venules in their investment of expansile lung tissue. From Plate II, Macklin, C. C., Trans. Roy. Soc. of Can., v. 39, Sect. 5, 3rd Ser., 1945. Rabbit A. S76-6R-2RM-9. 10 u, H. and E. X50. V, V_1, V_2 = venules; as = alveolar sacs. The arterioles, arteries and veins are similarly invested. The fine wrinkling of the walls is the result of technic.

Similarly in expanding they would not encroach upon the lumen of the venule, but rather would tend to draw away from it. This dilatational movement is not as easy to grasp as is the elongational one, but a study of v_2 and its setting of air sacs will help to make it plain. The expansion of this ring, or better, sleeve, aided by the blood pressure, would enlarge the vascular calibre. The same sort of radial pull on the part of the lung stroma is exerted upon the arteries as well as the veins (Macklin, 1945a). Some years ago it was shown to occur for the air tubes (Macklin, 1936). It is well to note that it is the normal lung which is being here considered, for in abnormal conditions there occur alterations in the mechanisms involved. The collective action of these motor sleeves of the blood vessels is shown in figure 2. In elongation the direction of shift of the long axis of the vessel is toward the narrower end.

Roentgenography of the living: It has long been known that corresponding bronchovascular rays are considerably longer in the inspiratory than in the expiratory stage, as shown by their measurements in comparative roentgenograms, one of which was taken in full expiration and the other immediately thereafter in full inspiration, from the same healthy young subject in the same position. This is a convincing demonstration, as far as it goes, that there is inspiratory lengthening of the roentgenographically apprehensible parts of the arteries and veins; but this technic has not been well adapted to the display of inspiratory widening. More information has been gained for the bronchi by this method, particularly when their shadows are intensified by iodized oil. The

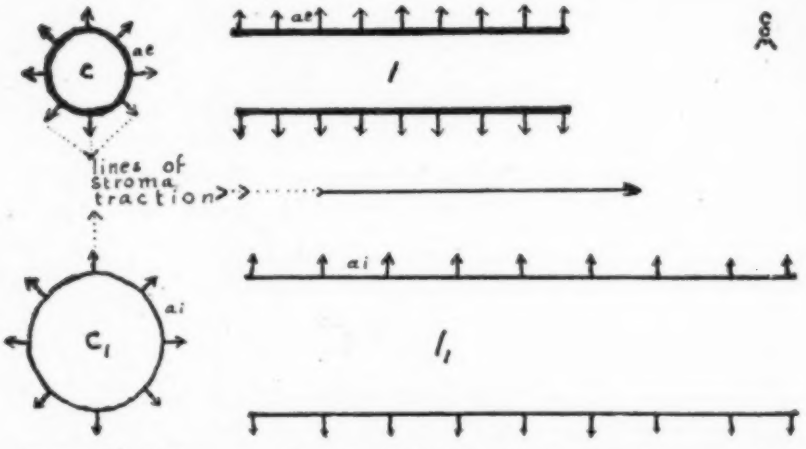

FIGURE 2—A diagram to show the expansile and extensile action on a pulmonary blood vessel of inflation of the surrounding alveoli. *c* and *l* show the same vessel in cross and longitudinal section in the phase of *expiration*: and c_1 and l_1 give the same views of the same vessel in the phase of *inspiration*. *ae* is an alveolus in expiration, and *ai* is the same in inspiration. Only the bases and part of the side walls of the alveoli are seen.

technic described by Taylor in his paper at this meeting on "Clinical application of angiocardiography" would seem to hold out invitingly the promise of clearer visualization of the shadows of the pulmonary blood vessels in the contrasted phases of full expiration and full inspiration.

Roentgenography of the dead: More clear-cut evidence for elongational and dilatational movements of these vessels is obtained when the lumina are filled with a fluid radio-opaque mass* which will not run through the capillaries to cloud the picture under the conditions of the experiment. If in two equivalent animals the pulmonary artery of one is filled in collapse and of the other in moderate inflation, we see strikingly in the two radiograms that the vessels of the latter are not only considerably longer but also are generally wider than are those of the former, which speaks emphatically for the exertion of a radial traction upon the vessel walls by the lung stroma in the inflated condition (Macklin, 1945b). Even more dramatic is the result when a single fresh lung is used for the two contrasting phases (Macklin, 1946). Here the pulmonary arterial system is first filled with a radio-opaque fluid mass introduced from a reservoir *via* rubber tubing and a cannula tied into the stem of the artery just beyond the heart, whereupon an x-ray picture is immediately taken to show the arterial shadow in the collapsed lung. The lung is then inflated by mouth through a cannula in the trachea, at which the interesting reaction is beheld of the fluid in the reservoir *falling* as air is going into the lung, so indicating without doubt that the capacity of the pulmonary arterial system is being enlarged. New fluid is added to the reservoir during this time to keep the pressure constant. When as much fluid has entered the artery as it will take, with the pressure level approximating that in the collapsed lung, a second x-ray picture is taken. This shows that the various branches of the artery are longer, and usually wider, than in the collapsed state.

While these experiments do not duplicate the phases of expiration and inspiration, and the conditions are far from those of the living healthy animal, yet they and others of the same type serve to show that when the air in the sacs is increased even under moderate pressure there is created *more room* within the arteries and veins. It is found, however, that when the lung is overinflated, that is blown up till the pleura is quite taut, it is possible,

*Synthetic latex, known as "neoprene" was obtained from American Anode, Inc., Akron, Ohio. It was made opaque by the addition of 10 to 20 per cent thorotrast (Heyden Chemical Corporation, New York). Another radio-opaque fluid mass was bismuth oxychloride (BiOCl) 5 per cent and gum acacia 10 per cent in water. These show the arterial or venous tree, according to which trunk is injected, without capillary clouding, at pressures of about 18 inches.

by increasing the air-pressure, actually to force fluid *higher* in the reservoir. X-ray pictures taken under such extreme air-pressure show the vessels much elongated but also narrowed. This state of affairs, of course, is not that of normal breathing, but rather is analogous to artificial respiration in the closed chest by tracheal insufflation with overly high pressure.

In other similar experiments the pulmonary vein system was injected in the same way in collapse and inflation, and x-rayed in these states; and the results were essentially like those of the artery. In another case both arterial and venous systems were injected in the same lung, and x-rayed, with results of the same sort. On re-collapsing such a lung with filled arterial or venous tree, and radiographing it, the shadow showed that the vessels had returned almost to the state of original collapse, most of the extra amount which had been taken up in inflation having been evacuated.

Volumetrics: The filling experiments mentioned suggested others (Macklin, 1946). Diluted latex* was used to fill two vertical calibrated reservoirs of glass tubing, one of which led into the pulmonary artery and the other into the pulmonary veins *via* the left atrium, of a dog heart-lung preparation.** The two vascular trees of the collapsed lung were first filled by gravity and the levels of the latex in the two tubes photographically recorded. The lung was then blown up by mouth through a tube in the trachea, during which process the latex fell fairly evenly in both tubes. Upon release of the air from the lung the latex rose in the tubes. Quantitative results were obtained. Since the air-pressure used was above that of the atmosphere the demonstration of inflational vascular enlargement was even more striking than it would have been had the conditions been those of the living thorax; for with the higher pressure in the air sacs it might have been expected that the latex would have been forced out of the vessels, causing the level to rise in the tubes during the inflation, but the opposite result was obtained. This experiment, which was repeated several times, gave a graphic illustration of the enlarging power of the expanding lung upon the capacity of the pulmonary arteries and veins. To make the demonstration even more dramatic a motion picture was taken showing the

*The same latex (see footnote, p. 537), diluted with 50 per cent distilled water, gave the best results. It was felt that the capillary net was excluded. When saline solution was used in the burettes it was felt that the capillary net was included (see Macklin, 1946).

**The pulmonary blood vessels were perfused with saline solution prior to being filled for roentgenograms or volumetric studies, and in many cases sodium nitrite was included to relax the muscle. When the sodium nitrite was omitted a slenderer vascular shadow was cast in the x-rays.

descent of the latex in the tubes while the lung was being blown up and its ascent during collapse. As before, if the pressure became too high during the inflation process, so that the pleura would no longer yield, the latex rose in the tubes, showing that it was possible to override the vascular enlarging mechanism by inhibiting the action of the stroma-pull.

But perhaps the most striking of all exhibitions of the enlarging effect of expanding lung tissue upon the arteries and veins was obtained when the lung was mounted on a platform which could be raised and lowered. After the arteries and veins had been filled with latex by gravity pressure the collapsed lung was raised until the cannulae leading into these vessels were on the same level as the latex in the tubes. Now when the lung was inflated the latex was actually drawn into the enlarged arteries and veins *against gravity*. The descent of the latex in the tubes was as much as 5 cm.

In this series of experiments the comparison was made of the fully collapsed with the inflated lung; but all the way along it was possible to compare vascular volumes of states of lesser inflation with those of greater, so obtaining data more nearly like those of the living, breathing lung; and always in passing from the lesser to the greater degree of inflation there was an enlargement of the capacity both in arteries and veins. It seems reasonable to conclude that in inspiration the pulmonary vascular volume is increased. In one case this increase was found to be about 33-1/3 per cent over the volume in the collapsed lung. A difficulty in these experiments was encountered from the rupturing of alveoli and leakage of air into the interstitial tissue, thus engendering pulmonic interstitial emphysema (Macklin, 1940; Macklin and Macklin, 1943; 1944). Masses of bubbles were often found at the end of the experiment under the pleura at the root of the lung. Another difficulty was the appearance of air emboli, particularly in the arteries, doubtless from invasion of the smaller blood vessels.

(B) *Expiratory shortening and narrowing.*

The converse phase in the arteries and veins is a shortening and narrowing movement. By studying the histology of the part it is evident that the diminution in size of the sleeve of air sacs around any given length of the living artery or vein would be attended by a contraction of the enclosed vessel in length and width, and the behavior of the two great vascular trees is a replica, on an enlarged and complex scale, of what goes on in any fraction thereof. When the tension of the enveloping stroma lessens, the intrinsic elastic tissue of the vascular walls is able to recoil. Since so much of this elastic tissue is in the form of fibers running lengthwise in the walls, the shortening effect of

its recoil is material. Many elastic fibers, too, run in spirals and these, on shortening, narrow the vessels. Such movements go on even against the pressure of the blood within. It may be that the smooth muscle plays a part in this narrowing process (Macklin, 1945a) just as it has been thought to do in the bronchial tree in the respiratory cycle (Macklin, 1929). It may be noted that workers can agree upon little if anything definite for the pulmonary vasomotor mechanism to do (Cournand, 1947) and it may be that it is adjusted to the respiratory rhythm. Then too, the air pressure bearing down upon the vessels in expiration is greater than that of the atmosphere, and on this point something will be said later on. We have noted how, in the filling experiments, the lung could recoil so forcibly as to raise the level of the fluid in the reservoirs considerably, and much of this work is attributable to the contraction of the elastic fibers of the air sacs and bronchial tree, aiding those of the vessels.

(C) *The capillary net and its implication in respiratory movements.*

In this rhythmic rise and fall of blood volume in the pulmonary vasculature the capillary net must share, and this it must do passively, motivated by environmental pressure fluctuations, for its loops protrude freely into the air spaces and its walls are not acted upon by the traction of the stroma in inspiration. With inspiratory lowering of environmental pressure the capillaries swell and with its expiratory elevation they shrink. Of similar order is the behavior of the precapillary arterioles and postcapillary venules. Looked at in "horizontal" view, the length of any given capillary is but an infinitesimal part of the total length of the pulmonary vascular bridge. The direct traction of the expansile tissue on the arteries and veins probably has the effect of protecting the capillaries from the bad effects of "negative pressure" by distributing the strain of expansion over the whole circuit rather than having it bear down unduly upon the capillaries as would be the case if the vascular system were "lung-free" (Macklin, 1945a).

D. *Pulmonic blood-pumping movements in quiet breathing.*

From what has been said it is concluded that the capacity of the entire pulmonary vascular bridge—arteries, capillaries and veins—is increased in inspiration and decreased in expiration; and that the constant repetition of this sequence has the effect of assisting the onward flow of the blood. It might be designated as a series of alternating systoles and diastoles. In expiration the vascular environmental pressure is greater than that of the atmosphere. There is then a pneumatic cushion pressing upon

the vessels. The blood cannot go back into the right ventricle on account of the pulmonary semilunar valve, and must go on toward and into the left atrium. This pump action, even at minimal level, would seem to function as a cardiac adjuvant. The site of greatest degree of volume change is doubtless the massed peripheral parts of the arterial and venous trees where the channels are smaller, thinner-walled and more numerous. This, then, would seem to be the most important part of the "pump." It is fortunate that the arteries and veins are so yielding, for if they were completely rigid we could have no breathing action of the lung at all. Fortunately pulmonary arteriosclerosis (Macklin and Macklin, 1942) never becomes so extreme as to produce such a catastrophe.

(E) *The augmented pump.*

But the bridge is able to increase its capacity enormously in response to demand, and the contemplation of this protective and accommodational reaction in the young athlete in violent and prolonged physical exertion is very arresting. Without an enormous enlargement of the traffic capacity of the bridge there would be a jam at the approaches, *i.e.*, right heart dilatation and systemic venous congestion. The traffic is speeded up of course; but in addition the effective value of the thoroughfare is multiplied by the widening of the channels. To get the greatest width the lungs must be held as fully expanded as possible, and this means that the chest tends to be operated at or near its maximal degree of expansion. If we look at a young man coming in at the end of a gruelling race we find him with head up and chest high and full, with even the accessory muscles of inspiration taut and pulling open the thorax as much as possible. In strenuous exercise the systemic circulation, of course, is vastly increased, and this means that the pulmonary circulation must be increased in like degree. The two go together. There can be no efficient augmentation of the systemic circulation without an equal augmentation of the pulmonary circulation. So the pulmonic bridge must bear just as much increase of traffic as that experienced by the entire systemic circulation at this quickened tempo, for the two circuits are really one blood course. It is fortunate that this pulmonary suspension bridge can have its traffic lanes pulled wider by innumerable guy-ropes held taut by the expanded air spaces. Respiration under these circumstances is rapid and forcible, but it is at the upper level of chest capacity. Of course it is commonly said that this extreme chest expansion is for the purpose of getting as much air as possible into the lungs, and although no one would gainsay the essential truth of this statement, it is equally true that it is for the purpose of getting as much blood as possible

across the bridge in any given unit of time, not only for the purpose of picking up oxygen on the way but of keeping the movement of blood through the lungs equal to that through the rest of the body in order to avoid a "pile-up" at the bridge entrance. The chemical and nervous controls involved in this pump action are too complex to be discussed here. The general body circulation—the systemic bridge—controls the situation, carrying blood in great quantities and delivering much carbon dioxide into it to stimulate the respiratory center. It is interesting that the heart beats faster during inspiration than it does during expiration (sinus arrhythmia).

II. *Implication of the Bridge in Operative Procedures.*

(A) *A multipartite bridge.*

We have been so much concerned with the physiological action of our bridge that we have not taken time to consider its anatomy; and when we do so we find that it is really two main bridges, one for each lung, and furthermore that each of these is subdivided into laneways for the lobes, which again are subdivided into smaller laneways for the lobules. Local alterations in the blood traffic follow certain medical and surgical operative procedures, producing hypoemia in some regions and hyperemia in others.

(B) *Compensatory hyperemia.*

So far we have been concerned with the variable capacity of the entire bridge in the healthy person, particularly the young athlete. Now we enter the domain of pathology and look for a moment at the behavior of the parts of the bridge which remain after a section of it has been removed. Always the surviving portion of the bridge has to carry the *entire traffic;* not only its own share but the share of the part which has been removed, be it a lobe, two lobes, an entire lung, or even one lung and part of another. That is not to say that the task is done as well as before under all conditions, and it would seem that the functional reserve is cut down; but it is remarkable how well the bridge functions in its reduced dimensions. It makes up for the loss of traffic lanes by the widening of those that remain, and in this widening process compensatory emphysema (alveolar ectasia) and compensatory hyperemia go hand in hand. As always, the pull of the stroma, actuated by air pressure, widens the bridge (Macklin, 1945b), and enables it to carry the increased load. The enlargement of the arteries and veins so brought about probably prevents the development of "Factor B" and the supervention of pulmonary interstitial emphysema (Macklin and Macklin, 1944). I have been told (W. E. Adams[14]) that healthy dogs can survive with only some 15 per cent

of their lung substance remaining after operation, and that means that 15 per cent of the bridge is carrying 100 per cent of the blood traffic.

This amazing ability of the bridge to compensate for the loss of parts of itself makes much of modern thoracic surgery possible. Who would dare to take out a lung or even a lobe if the remaining pulmonary blood streambed did not conveniently adapt itself to carrying without obstruction the share of the parts removed? Imagine what would happen to the patient if, after removal of a lung, the vessels of the other lung refused to carry the extra load! Fortunate indeed is it that the bridge has such great reserve capacity in its various parts, for this may be utilized by physicians, particularly chest physicians.

(C) *Relaxation therapy and accompanying hypoemia.*

But sections of the bridge may be altered in respect of their blood flow in ways other than by extirpation of part of the lung, and one of these is therapeutic collapse of more or less of the lung substance. By pneumothorax, phrenic paralysis or pneumoperitoneum the volume of an area of lung is reduced, as in the treatment of pulmonary tuberculosis. This has the effect of diminishing or abolishing the pull of the stroma upon the arteries and veins of the part. These vessels undergo shortening and narrowing, and the flow of blood in them is accordingly reduced. It is prudent for me to leave to the clinicians and pathologists the explanation of how relaxation therapy improves the local conditions and so benefits the patient. Certain it is that with relaxation comes rest, and rest for the diseased part often means opportunity for repair. We do not know as much about the metabolism of elastic tissue as we should; but it seems reasonable to assume that the constant extension and contraction of elastic fibers is attended by wear and tear which must be made good if the part is to continue to function well; and that the relief from these movements in a part harassed by disease would be beneficial. Condensation of the tissues means closing the ranks of the defending and repairing cells, so giving them a better chance, and there remains enough blood supply to meet their needs. Danger of hemorrhage is reduced and elimination of exudate favored. Then, too, it has been pointed out (Banyai, 1946) that the bacillus tuberculosis, being an aerobe, is put at a disadvantage in a therapeutically relaxed part of the lung because of local diminution of available oxygen resulting from hypoemia (hyphemia, hypoema). So all things work together for the good of the patient and the discomfiture of the invaders. With the reduction of the pulmonary blood flow in the relaxed part there is associated an augmentation

of the flow in other healthier parts, enabling them to assume larger duties. The nutritional (bronchial) blood supply is conserved in the relaxed part, and the healing of local tuberculous lesions favored. The paper of Barach *et al.* at this meeting on "Disappearance of cavity in the lung immobilizing chambers in nine patients with pulmonary tuberculosis" describes what appears to be a generalized relative collapse, or universal hypoinflation, of the unmoving lungs. In such a situation the pulmonary arteries and veins would not be subjected to inspiratory stroma pull, and the pump action would be abolished. This state in these vessels would seem to be the antithesis of that in the lungs of the strenuously exercising athlete, where they are fully opened up and showing the pump action in a marked degree.

III. *Deterioration of the Bridge.*

The pulmonary vascular bridge can deteriorate as a result of age or disease, and malfunction ensue. This change can be intrinsic, as seen in arterio-sclerosis, which would not only interfere with functional elongation and widening, but also with the contractile mechanism of the vessel walls. So the pumping movement would be curtailed. Consideration of the various aspects of extrinsic deterioration would take us too far afield for the purposes of this paper. It is an intriguing subject. The age changes alone (Macklin and Macklin, 1942) are very important. Bronchosclerosis, medical emphysema, fibrosis from many causes and pulmonary tuberculosis are among some of the many conditions which hamper the function of the bridge. Lobar pneumonia offers an alluring opportunity for the study of the impaired bridge, for the arteries and veins, on account of the expansion of the air-spaces with exudate, are elongated and possibly widened; but there could be no pump action, and the lack of this may help to explain the impaired circulation in the affected part. Even at that, the diseased lobe would act as a robber of pulmonic blood, contributing nothing of value and taxing the right heart while adding toxins to the body. Since the part tends to retain its volume to a great extent if not completely, the advantages of compensatory emphysema and hyperemia in more normal parts are difficult of attainment. Different is the situation in massive collapse, and to a lesser degree in any atelectatic area, for here, with the slackening of stroma traction and the ensuing local hypoemia, there can occur a compensatory hyperemia in other parts where compensatory emphysema increases the stroma pull and so enlarges the capacity of the bridge. There is a limit to the amount of space reduction which can occur in the unaltered thorax and when one region of lung becomes shrunken there is a tendency for other parts to swell with air to

fill the space, and we see this tendency exemplified in massive collapse with its blood shunt to other parts of the lung which have become hyperexpanded. Much more could be said under this heading, as in the consideration of bridge deterioration in the various heart derangements, and in a host of constitutional disorders; or in the strictly local accident of embolism or thrombosis with supervening infarction. Master's paper at this meeting on "Pulmonary embolism as a cause of acute coronary insufficiency" indicates that sudden interruption of pulmonary bridge traffic leads to diminution of systemic bridge traffic, with consequent aortic and coronary insufficiency, causing myocardial anoxia and ischemia. Anemia of the coronary system indeed would be apt to follow interruption of pulmonary bridge traffic of gradual, as well as sudden, onset. Pulmonic interstitial emphysema, by pressing on the arterial and venous walls, impedes bridge traffic. Withdrawal of air has given relief (Macklin and Macklin, 1944).

IV. *Assistance to the Bridge.*

Fortunately in health the bridge operates automatically. Its chemical and neuronal controls carry on so smoothly that there is no need to worry. Automaticity rules. We have noted how it can overcome seemingly impossible difficulties and handle prodigious masses of blood as in the Marathon runner. Yet even in the case of Pheidippides it seems likely that there was a fatal traffic jam because he did not give heed to the care of his bridge but felt obliged to make a speech instead of to keep his pulmonary traffic lanes wide open and the "Brustorgan" working. We of older years and poorer physical condition should take good care of our pulmonary bridges. If we have to run for a car or climb a long stair or shovel snow or carry out heavy loads of ashes from the furnace we should, if out of breath, stop and fill our lungs full, breathing hard at the top level of chest capacity; in short we should imitate the athlete. We should advise our patients to do this. Thus we may prevent right heart over-dilatation and coronary anemia; may avoid tragedy. Better still, if need be, we should lie down on the back, with our knees pulled up to give the diaphragm free scope, and in that position, with the blood stream horizontal, relieve the congestion at the bridge approaches by hard breathing with the thorax full. So will we save our hearts—our lives. Physician, look to your bridge!

SUMMARY

The lung tissue has a synergic action on the pulmonary arteries and veins which are interwoven in it, and particularly on those of smaller size, whereby their volume is increased in inflation

and decreased in deflation. This rhythmic volume change forms what has been called a "lung pulse," which is experienced passively by the capillary bed, and is most marked in vigorous exercise. Thus the lung, motivated by the musculature of the chest walls and diaphragm, has the effect of an accessory heart. This lung pump, when needed, should be kept working at full capacity. Sudden reduction of its action, as in runners or oarsmen, after a gruelling race, would have serious, even disastrous consequences. When resting in the ascent of a long stair or after other marked exertion one should breathe forcibly with the lungs well filled, for in this way blood is best moved across the "thoracic bridge" and congestion of the right heart and coronary anemia prevented. Dilatational action is by a direct pull of the lung stroma upon the arterial and venous walls during inspiration, which tends to increase the length and width of these vessels. The peripheral resistance in the pulmonary circulation is thus lowered by enlargement of the streambed due to outside assistance, and the right heart is aided. In the vascular volume decrease of expiration there is, following release of the stroma tension, a recoil of the elastic fibers of the vessel walls, leading to shortening and narrowing of the vessels. Histological, roentgenological and volumetric evidence is advanced in support of these conclusions. Advanced age, alveolar emphysema and other conditions decrease the efficiency of this lung pump.

Artificial relaxation of the lung, by abolishing stroma pull on the arteries and veins, diminishes the effective caliber of the pulmonary vasculature, so reducing the physiological blood flow while maintaining the nutritional flow in the bronchial vessels. There would probably develop in the collapsed lung region a relative tissue anoxemia and carbon dioxide increase, thus providing conditions inimical to the growth of the tubercle bacillus, while maintaining sufficient vitality in the tissues to insure regeneration and healing.

RESUMEN

El tejido pulmonar ejerce una acción sinérgica sobre las arterias y venas pulmonares que están entrelazadas en él, y particularmente sobre las de pequeño tamaño, por medio del cual sus volúmenes aumentan durante la inflación y disminuyen durante la desinflación. Esta alteración rítmica del volumen forma lo que se ha llamado un "pulso pulmonar," que lo sufre pasivamente la red capilar y que es más marcado durante el ejercicio vigoroso. De manera que el pulmón, motivado por la musculatura de las paredes del tórax y el diafragma, hace el efecto de un corazón accesorio. Esta bomba pulmonar, cuando se la necesita, debe man-

tenerse funcionando en su capacidad total. La reducción repentina de su acción, como en corredores o en remeros, después de una carrera agotadora, tendría consecuencias graves y aún desastrosas. Cuando se descansa, durante la subida de una escalera larga o después de otro esfuerzo marcado, debe uno respirar forzosamente y llenarse bien los pulmones, porque de esta manera se mueve mejor la sangre a través del "puente torácico" y se evita la congestión del corazón derecho y la anemia coronaria. La acción dilatadora se debe a que el estroma pulmonar ejerce un tirón directo sobre las paredes de las arterias y venas durante la inspiración, lo que tiende a aumentar la longitud y anchura de esos vasos. La resistencia periférica en la circulación pulmonar queda disminuída debido al ensanchamiento de la red vascular causada por la ayuda externa, y esto asiste al corazón derecho. Cuando disminuye la tensión del estroma durante la fase expiratoria, decrece el volumen vascular debido al retroceso de las fibras elásticas de las paredes de los vasos, lo que causa que se acorten y angosten estos vasos. Se presentan datos histológicos, roentgenológicos y volumétricos para apoyar estas conclusiones. La edad avanzada, el enfisema alveolar y otras condiciones disminuyen la eficiencia de esta bomba pulmonar.

La dilatación artificial del pulmón destruye la acción del estroma sobre las arterias y venas y disminuye así el calibre efectivo de la red vascular del pulmón, reduciendo de esta manera la circulación fisiológica pero manteniendo la circulación nutritiva en los vasos bronquiales. En la región colapsada del pulmón probablemente se desarrolla una relativa anoxemia y aumento del anhídrido carbónico de los tejidos, lo que produce condiciones perjudiciales al crecimiento del bacilo tuberculoso, pero mantiene suficiente vitalidad en los tejidos para asegurar la regeneración y cicatrización.

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Discussion

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Professor Macklin has presented clear cut evidence of a fundamental concept. That the physiological factors that play a part in producing bronchial changes are applicable to the pulmonary vessels is a concept not difficult to accept.

To date, there have been no studies of this type made on humans. Fortunately I found a patient with emphysema, asthma, and dilated pulmonary vessels. Inspiration and expiration films were made and changes of the right hilum were measured. A gross change in maximum length and width of the right hilum was easily demonstrated. There was a thirteen per cent increase in area on inspiration.

The areas of two pulmonary arteries increased on inspiration; a large one increased fourteen per cent, and a smaller one increased twenty-four per cent.

These findings agree with Dr. Macklin's. The application of a suitable technique in kymography and angiography would be of great interest in this work.

Discussion

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The pulmonary circulation and its possible relationship to the pathogenesis of certain diseases is a subject which is not well understood. When it is, such questions as the mysterious preponderance of pulmonary tuberculosis in the apices, the greater frequency of hydrothorax on the right side, more common occurance of antemortem thrombosis of the pulmonary artery, the greater incidence of lower lobe tuberculosis on the right side and other equally curious conditions may become clear. Doctor Macklin has demonstrated that the pulmonary tissue has a pulling action on the vessels, and particularly on those of smaller size, whereby their volume is increased in inflation and decreased in deflation. This change of the volume which he has designated a "lung pulse" is most pronounced in vigorous exercise. The dilatational action is by a direct pull of the pulmonary parenchyma upon the walls of the vessels. The collapse therapy, by abolishing the pull on the vessels, diminished the caliber of the vessels, so reducing the volume of blood flow.

The postmortem arteriograms of the collapsed lungs demonstrated the shortening and narrowing of the lumens of the vessels. The quantitative measurements of the latex flowing through the models constructed after these arteriograms suggested that the minute volume of the blood through the vessels in the collapsed lung may be smaller than that in the normal lung at any one time, but the speed of the flow seems faster. Therefore, after a certain length of time, the total volume of blood flowing through the collapsed lung may be even greater than that through the non-collapsed lung.

The speed of blood flow seems faster in the left lung as shown by the determination of the velocity of the pleura to the tongue circulation time, which will appear in "Diseases of the Chest." The reason for this may be due to the greater massaging effect of the cardiac impact upon the left lung. The reason for the lesser frequency of hydrothorax on the left side may be due to faster blood flow through this lung by the reason of greater pulmonary passive movements.

Mr. Chairman. We hope that there will be more investigations on the physiology of the pulmonary circulation, a part of which was so clearly demonstrated by Doctor Macklin.

Silicosis as Viewed by an Internist*

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The presence of silicosis in man dates back to early times. According to Hale[1] it may be the earliest of all occupational diseases. This condition was first described by Hippocrates who noted it in certain metal grinders, but it has only been during the last 25 years that our knowledge of silicosis has been materially increased.

Not all persons exposed to silica dust develop silicosis. The following conditions influence the production of this disease: (1) Size of the silica particles. (2) Number of particles per cubic foot of air present at the breathing level. (3) Free or combined state of silica. (4) Duration of inhalation. (5) Humidity of working atmosphere. (6) Condition of the ciliated epithelium of the individual.

According to the Department of Labor[2] and U. S. Public Health Service[3] investigations, particles larger than 10 microns in size are not capable of producing pulmonary fibrosis, and are not found beyond this size on microscopic examinations of the lung. The greatest harm is produced by those between 1 and 3 microns.

The National Silicosis Conference holds that exposure to a concentration up to 5 million particles per cubic foot of dust composed of 70 to 80 per cent silica is without danger, but over this concentration there is the possibility of developing silicosis.

In areas such as Colorado where the silica particles in the dust average about 35 per cent, 10 million particles per cubic foot may be present without development of silicosis, when one is exposed for ten years; however, when the concentration reaches 20 million particles, silicosis may appear after ten years of exposure. The higher the concentration the more rapid the disease is liable to develop.

Cummings[4] states that moisture is helpful in the prevention of dust and has reduced the incidence of silicosis among workers, but others have reported an increasing incidence of tuberculous infection among workers in mines with high humidity. He feels that adequate ventilation and humidity within normal limits is preferable.

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The ciliated epithelium of the trachea and bronchi prevents many of the fine particles of silica from entering the lungs. The condition of the ciliated epithelium probably accounts for the varied degrees of development of silicosis in individuals working under the same conditions. Previous infection, causing destruction or decreased motility of the cilia in certain individuals, greatly alters the ability of the system to remove the particles of inhaled dust. The work of Barclay,[5] et al, on ciliary movement is convincing.

Chronic irritation of the air passages results in replacement of ciliated epithelium by goblet cells and increased production of mucus by the buried glands. The absence of ciliated epithelium which normally removes dust and the excess mucus facilitate the downward movement of foreign material.

When dust passes the protective cilia and reaches the pulmonary alveoli, the phagocytes emerge from the capillary walls and ingest the particles. When the phagocyte becomes laden with silica particles it enters the adjacent lymph spaces and passes toward the lung hilum. A trail of collagen forms after some of these phagocytes pass which is thought to be produced by the stimulated but normal adjacent tissues. Early in the exposure to silica some of the phagocytes pursue a simple course into the blood stream. Later fibrous tissue prevents easy passage of the phagocytes and their load of irritating silica is deposited, resulting in more fibrosis. In time, the nodules attain such size as to cast x-ray shadows. As these nodules become larger they coalesce, and offer more obstruction to the x-ray.[6] The development of demonstrable silicosis may require from 1½ to 30 years.

Symptoms and Physical Signs

Symptoms and physical signs may be lacking even in an advanced case of silicosis; therefore careful history taking is of the greatest importance in the detection of this disease. When present, the most characteristic symptom is dyspnea. At first there may be little or no cough, but in the later stages cough and expectoration usually are present, particularly in the morning. As the case advances there is a gradual failing in health and strength and vague gastrointestinal disturbances appear. In uncomplicated cases there is usually no fever unless a tuberculous process or other infection develops.

Physical signs in the chest are usually those found in a bronchitis, along with signs of emphysema, however many cases have no physical signs whatsoever. The roentgenogram is an essential medium in the diagnosis of silicosis.

Classification

Many excellent classifications have been given for this disease by such authorities as Pancoast,[7] Pendergrass,[8] Gardner[9] and Sampson.[10] For this discussion the following classification of Garland[11] is followed:

1) Incipient Type: Hilar shadows wider and denser than normal; perihilar pulmonary tree markings wider than normal.

2) Interstitial Type: Hilar shadows wider and denser than normal, slight or moderate diffuse thickening of the pulmonary tree markings, associated with faint haze in the middle or lower thirds of the lungs in early cases; extensive thickening in well established cases. Limitation of diaphragmatic excursion (varying from slight to marked).

3) Nodular Type: Hilar shadows wider and denser than normal; diffuse scattering of small, dense, discrete nodules throughout both lungs, especially in the middle thirds; apices and costophrenic angles usually free; limitation of diaphragmatic excursion.

4) Advanced (or Conglomerate Type): Hilar shadows less prominent than usual, diffuse thickening of pulmonary tree markings, and/or diffuse nodulations, small or large areas of density due to coalescence of fibrotic or nodular lesions; emphysema; limitation of diaphragmatic excursion; occasional cavitation; enlargement of the right side of the heart.

Conditions Simulating Silicosis

There are certain diseases which simulate silicosis when studied by x-ray; however, careful interpretation of the x-ray films combined with clinical information should rule out most of these conditions. In the incipient type of silicosis, passive congestion of the lung may be confusing. In the interstitial type, passive congestion of the lungs (cardiac), acute and chronic respiratory infections, polycythemia, metastatic malignancies, certain lymphoblastoma (infiltrative Hodgkin's disease) often confuse the picture. Miliary tuberculosis (Fig. 1A), metastatic malignancies, peri-arteritis nodosa (Fig. 1B), mycoses (Fig. 1C), and pulmonary arterial sclerosis (Fig. 1D), may often be confused with the nodular type.

Passive congestion of the lungs can be confused with silicosis if the individual is a worker in silica dust, as the following case illustrates:

Case 2, G.R.: This man came to our office February 3, 1943 complaining of extreme shortness of breath on exertion and had been coughing for several days. He was short of breath upon lying down. He gave a history of working in a quartz mine. Upon examination wheezing was heard throughout his chest, his temperature was 101 degrees and there was clubbing of the fingers. He was sent to Colorado General Hospital with a

diagnosis of bronchial pneumonia and cardiac failure, superimposed on silicosis. X-ray inspection revealed evidence of broncho-pneumonia and possible silicotic changes in both lungs (Fig. 2b).

The patient was placed on routine pneumonia and cardiac treatment, but rapidly failed and died on February 9, 1943. Autopsy revealed confluent bilateral bronchopneumonia, fibro-purulent pericarditis, dilatation and hypertrophy of the heart, subacute thrombosis of the descending

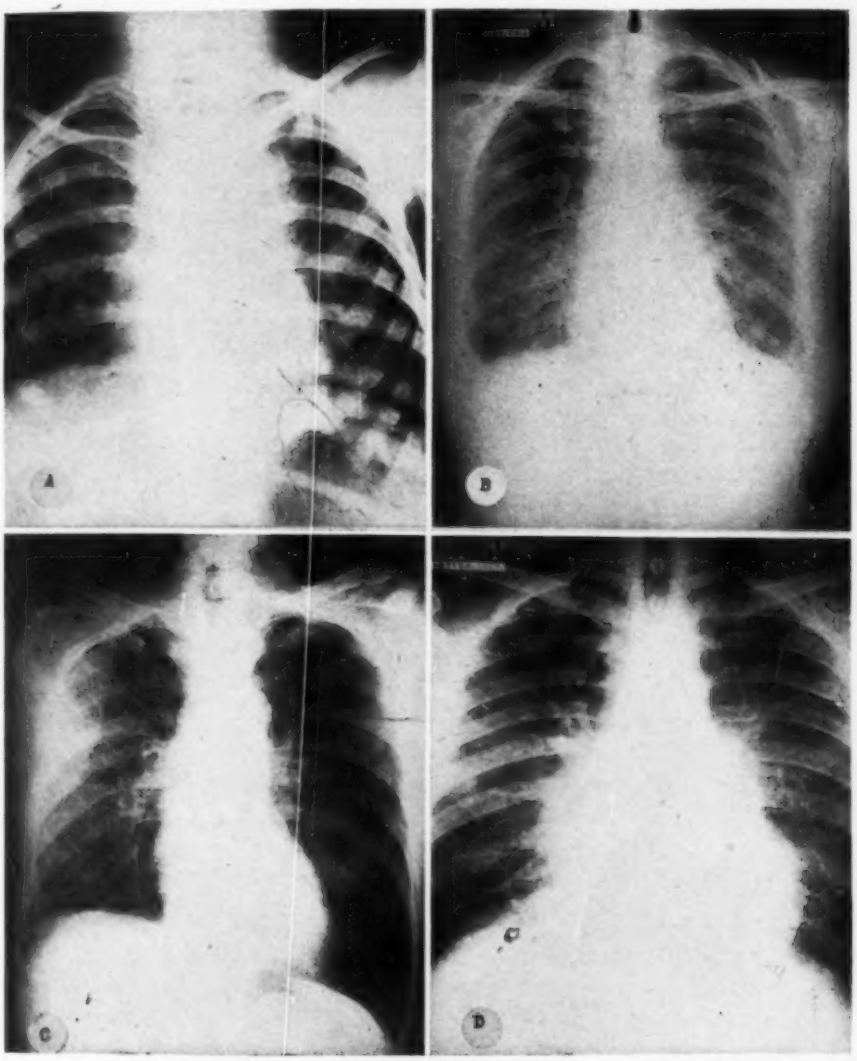

FIGURE 1: Cases showing conditions simulating silicosis

(A) Miliary tuberculosis: The tracheal bronchial rather than the hilum lymph nodes are enlarged. Bases not so clear and nodules more minute than in silicosis.—(B) Peri-arteritis nodosa: The bases are not so clear and the nodulation not as dense as in silicosis.—(C) Fungus infection—Blastomycoses: Less hilar involvement than in silicosis and asymmetrical involvement of the lung.—(D) Pulmonary arterial sclerosis: The trunk shadows are less prominent and the nodulation is finer and more granular. The bases are not so clear as in silicosis.

branch of the left coronary artery, fatty metamorphosis of the liver, cloudy swelling of the kidneys, moderate hydrothorax and ascites, but there was no evidence of silicosis.

We were able to obtain x-ray films taken June 13, 1942 which revealed little or no evidence of silicosis (Fig. 2a). The history of quartz mining mislead us in making a diagnosis of underlying silicosis. It later was learned that he worked for only 7 months for a mining company as a helper and oiler in the mill.

Symptoms due to polycythemia can be misleading, especially when this condition exists with mild silicosis.

Case 3, J.B.: This patient was first examined on March 24, 1938. He had fallen fifty feet in a mine shaft November 16, 1937 and was seriously injured. Our examination was made to determine whether his symptoms were due to this accident. He complained of weakness, constant headache, sleepiness during the day, shortness of breath, persistent cough and duskiness about the face which was more pronounced when he worked in the mountains. This condition had existed since 1928.

Examination revealed a well developed man with a cyanosis of the face and neck and slight exophthalmos. The eyes were congested. There was increased bronchial breathing throughout the chest. The liver was slightly enlarged. The hands and feet were cyanotic with clubbing of the fingers. Blood pressure was 130/80. The hemoglobin was 120 per cent and there were 8,220,000 red cells. The basal metabolic rate was +30 per cent. An electrocardiogram showed right preponderance and occasional right ventricular premature contraction.

On March 29, 1938 the x-ray report was as follows: "... early, second stage pneumoconiosis. The evidence of this is intensification of both hilum shadows and exaggeration of the lymphoid elements of both lungs. The bases of both lungs appear to be a little more clear than the remaining portion of the lung field" (Fig. 3a). "A study in the lateral oblique and postero-anterior view by stereoscopic examination does not disclose

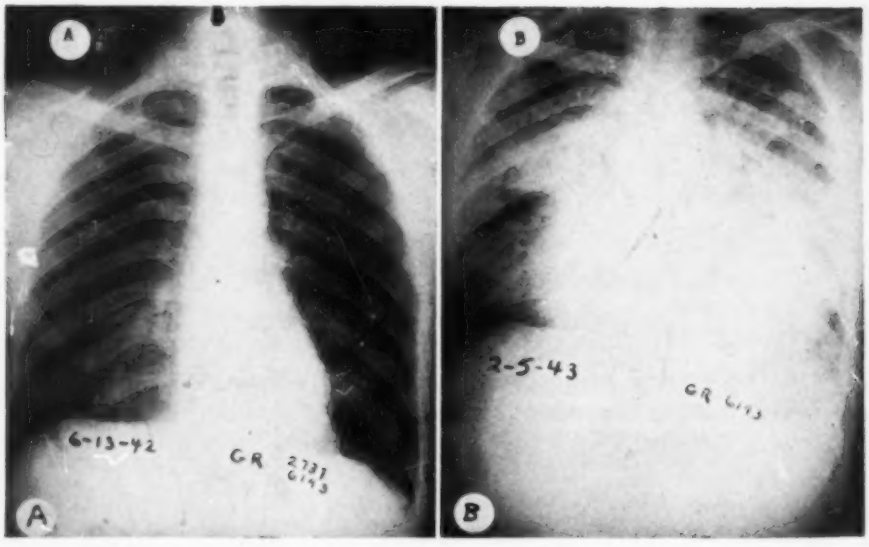

FIGURE 2, CASE 2: Passive congestion of the lungs simulating silicosis.

the roentgen signs of enlarged right or left pulmonary artery. A very slight enlargement cannot be excluded by x-ray examination" (Fig. 3b).

He was told he had polycythemia with possible Ayerza's disease superimposed upon silicosis. We obtained x-ray films and data taken on August 24, 1938 (Fig. 3c) and November 29, 1943 (Fig. 3d). The later film was similar to those taken in 1938 except that there was a marked increase in opacity to both hilum lymph node areas. This is an occasional finding in silicosis upon which we will later elaborate. He had not worked in silica dust for 6 years. His hemoglobin was 112 per cent red blood cell count 6,200,000. The electrocardiogram showed no right preponderance. This is probably due to the fact that the left ventricle is now slightly enlarged as well as the right.

This case illustrates that polycythemia and cardiac failure was the greatest disabling factor, even though silicosis was present.

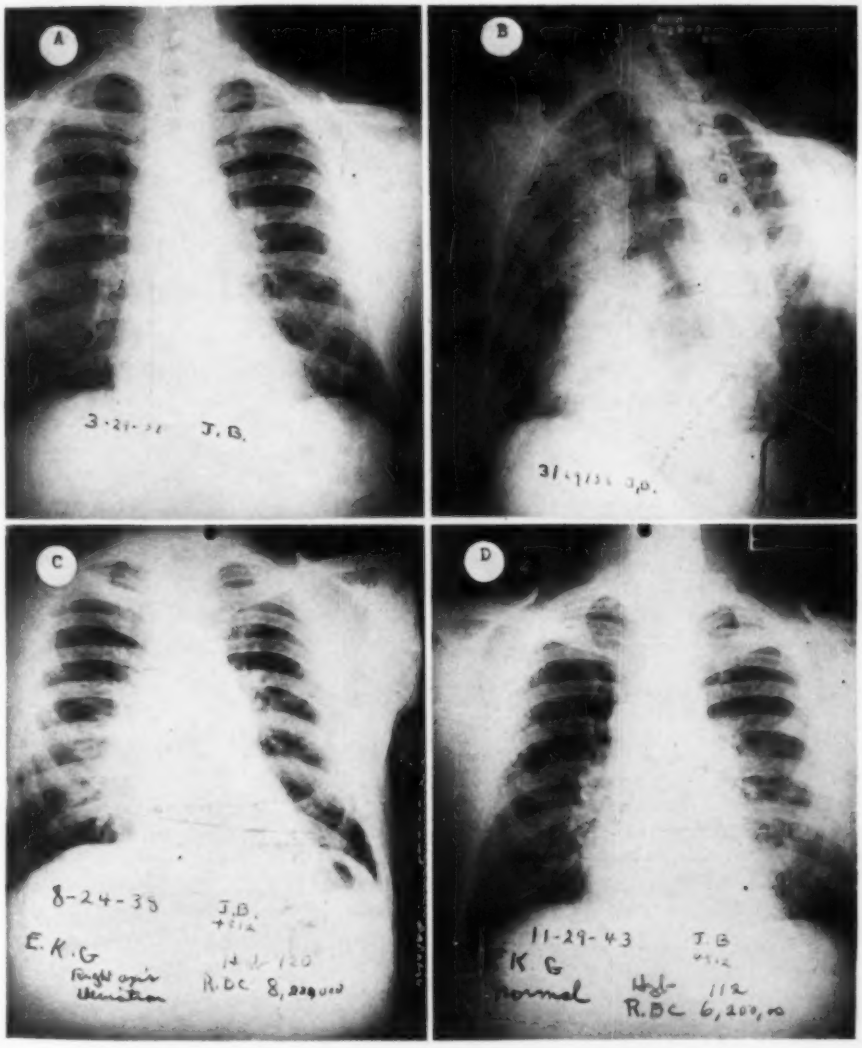

FIGURE 3, CASE 3: Polycythemia with silicosis.

Lung Tumors

Tumors of the lungs are often confused with silicosis; however, under certain conditions, the latter may be wrongly diagnosed as lung tumor, which is illustrated by the following case:

Case 4, F.M.: This man was injured May 23, 1932 by having an iron tripod fall against his chest and right shoulder. He was disabled for only six days and then returned to work in the quartz mine. On January 1, 1935 he stopped work because of shortness of breath, severe cough and weight loss which he thought were due to his 1932 accident. Our examination on June 18, 1935 revealed a rather sick appearing man, edentulous, with enlarged and infected tonsils. There was a small area of hyperresonance with slight decrease in breath sounds in the apex of the right lung. Below this there was a slight dullness. Inspiration sounds were rough and prolonged over the entire chest. The liver was slightly enlarged and a right inguinal hernia was present.

The x-ray report was as follows: "X-ray examination of the lung fields disclose confluent pneumoconiosis in both lungs. The right upper lobe region shows a more discrete density than other parts of the lung fields. This has been reported as a lung tumor elsewhere. A diagnosis of silicosis was made" (Fig. 4b). An x-ray film, taken elsewhere in April, 1935 showed somewhat the same condition except that in the right apex there was pneumothorax with a rather definite outline of the lung below (Fig. 4a). Otherwise the film was similar to that taken in June, 1935.

A diagnosis of lung tumor had been made elsewhere. The reason for this confusion possibly lies in the fact that a pneumothorax was present and compressed the silicotic lung giving it the appearance of a tumor. Autopsy in the spring of 1937 revealed no evidence of tumor but silicosis was present.

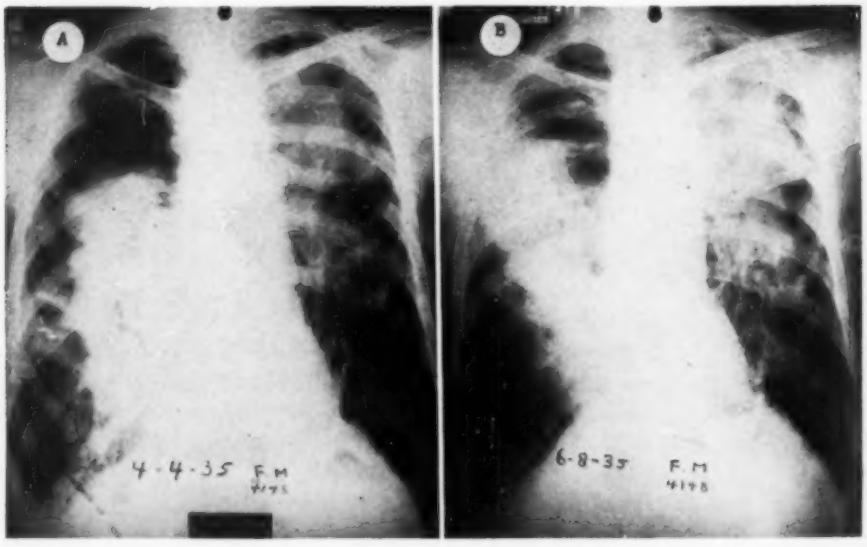

FIGURE 4, CASE 4: Silicosis simulating lung tumor.

Progress of Silicosis

The author has been interested for some time as to why some men working under similar conditions, develop the disease rapidly, and others show no definite progression.

Bamberger[12] has shown that among men working under the same conditions in mining operations at the same time, one develops only mild linear exaggerations while another presents far advanced nodular silicosis.

This condition probably is due to the inability of the cilia to protect against the dust invasion. The following cases illustrate the development of silicosis in men working under similar conditions at about the same time.

Case 5, C.G.P.: This man was born March 29, 1909 in Missouri and worked as a farmer until 1935 when he was employed for three months as a machine man in tunnel construction. Then for one year he worked in dam construction. In 1937 he began work as a quartz miner. On November 6, 1940 the first x-ray revealed slight changes (Fig. 5a) and by December 30, 1942 a definite nodular type of silicosis had developed (Fig. 5b). On January 21, 1943 he complained of dyspnea, pain over the heart, dizzy spells, persistent cough and rapid heart on exertion. There was a loss of appetite and weight. An electrocardiogram was normal except for right axis deviation.

Case 6, C.S.: This man was born on November 26, 1905 in Leadville, Colorado. From 1936 to 1942 he was employed as a quartz miner. The first x-ray inspection made on December 10, 1940 suggested the presence of silicosis (Fig. 6a). During 1941 he suffered a series of upper respiratory infections. By April 1, 1942 there were prominent linear markings with

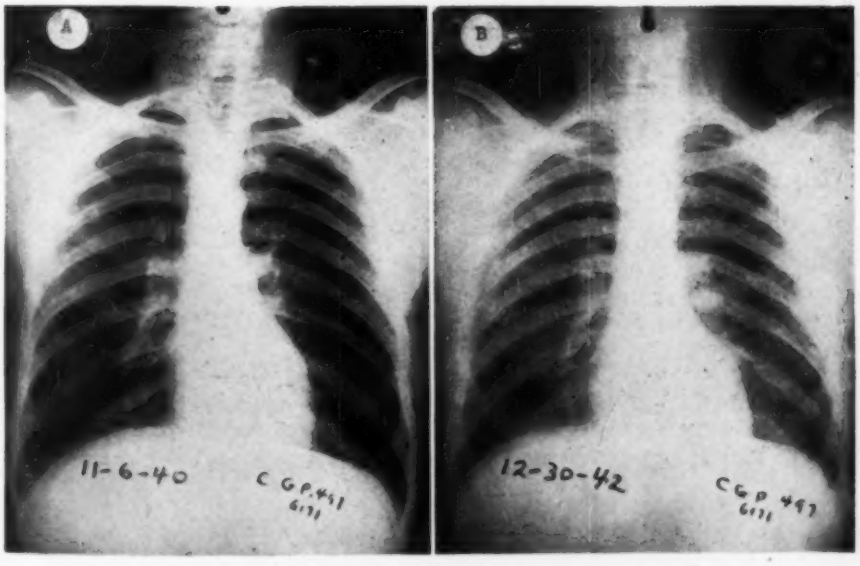

FIGURE 5, CASE 5: Progress of silicosis.

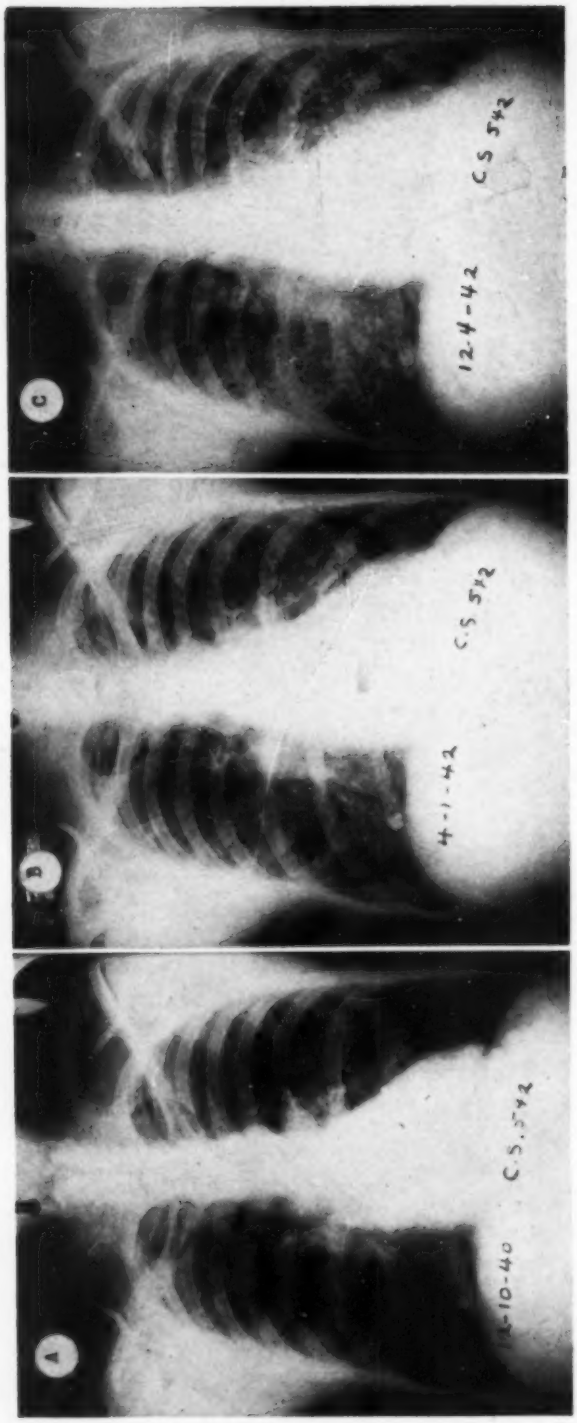

FIGURE 6, CASE 6: Progress of silicosis.

some areas of nodulation and increased hilar shadows (Fig. 6b). He was removed from the quartz dust and worked above ground. By December 4, 1942 there was evidence of advanced nodular silicosis, of the conglomerate form (Fig. 6c).

The above cases were working in the same mine under practically the same conditions including the time element, and it can be readily noted that they show different stages of silicosis. Other men working with them under the same conditions showed little or no evidence of silicosis. These cases afford evidence that infection and other conditions already discussed are factors in the variability of the development of silicosis.

Development of Silicosis After Removal from Dust

Usually the removal of the worker from exposure to silica dust checks further development of silicosis. There are some cases, who even though removed from inhalation of silica dust, continue to show signs of increased nodulation and fibrosis. Infection plays an important role as suggested by the following cases:

Case 7, J.D.W.: This man of 50 years was examined December 23, 1937. He had been a quartz miner since 14 years of age. In the previous eight weeks he had cough and raised sputum, lost 30 pounds and complained of dyspnea. There was increased bronchial breathing all over the chest, with some coarse rales to the right of the sternum. X-ray inspection on December 23, 1937 showed exaggeration of the linear markings and thickening of the pleura in the right upper interlobar fissure (Fig. 7a). X-ray inspection on February 2, 1938 showed increase of the linear markings since December. The lower portion of the right lung revealed definite inflammatory changes which apparently represented superimposed infection and increase of silicosis (Fig. 7b). This man had not worked in the mines for five months and yet there was evidence of advancement of the silicosis.

Case 7b illustrates advancement of silicosis, even though work in a quartz mine had been discontinued for 4 years. Dr. Berry kindly furnished the following data: This man of 36 years was a quartz miner for ten years. On May 5, 1943 there was evidence of slight nodulation and enlargement of the hilar shadows (Fig. 7c). The electrocardiogram showed evidence of pathological changes. He then was in the army until 1946. During this interval there was no history of exposure to silica dust. He was asymptomatic but on April 18, 1946 discrete nodulation was seen throughout both lungs (Fig. 7d).

In addition to the above cases some of our previously quoted cases showed the same phenomena (Case 3, Fig. 3 and Case 6, Fig. 6).

Cardiac and Tuberculous Complications

When silicosis reaches the advanced or massive conglomerate form most cases begin to complain of shortness of breath, especially in high altitudes, due to decreased respiratory reserve. Occas-

ionally cases of far advanced silicosis suffer little dyspnea but as a rule a compensatory polycythemia is found. In those cases that develop a cardiac condition right heart failure is the most common. Often before cardiac decompensation occurs the only sign present is an accentuated pulmonary second sound. In some of our cases right ventricular enlargement by x-ray and right axis deviation could be demonstrated before cardiac decompensation occurred. An occasional case has been found to have left ventricular hypertrophy but usually at autopsy, bilateral ventricular hypertrophy is present.

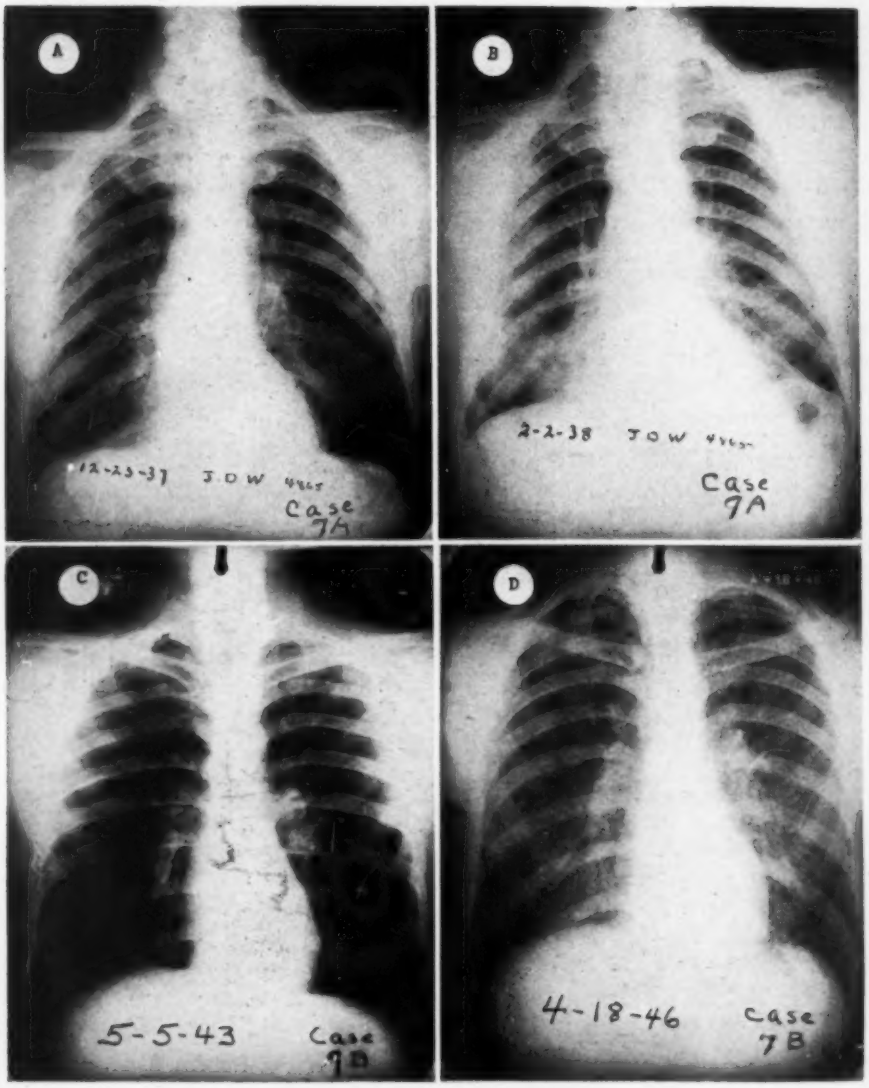

FIGURE 7, CASES 7A and 7B:
Showing development of silicosis after removal from dust.

According to Gardner[13] from 20 to 30 per cent of people with silicosis ultimately die from heart failure that comes on suddenly and terminates in a few months and most of the rest succumb to tuberculosis. However other workers, particularly Sander,[13a] have reported much lower figures. In Colorado it is our opinion that the percentage of cardiac deaths is slightly higher, and that of tuberculosis lower as a terminal event. This holds for those cases who have resided in Colorado most of their lives. We think this is also true of development of adult type tuberculosis in the non-silicotic individual, who resides in this area, as the climate, altitude, sunshine and lack of over-crowding, tend to lessen the liability of the development of the disease. This same contention was advanced by Henry Sewall[14] and contributed to by other workers.[15,16] Statistics are now being analyzed to test our theory but many factors interfere with making final conclusions. This observation may differ from other opinions due to the fact that the author confines his work to private practice and general hospitals rather than sanitoria that handle tuberculous cases only.

When tuberculosis does develop in the presence of silicosis we agree with Dayman[17] that the constitutional reactions are not so pronounced as when tuberculosis alone is present. If a diagnosis is made early, the tuberculous lesion usually clears if proper care is instituted, as the following case illustrates:

Case 8, J.D.: Born in Nebraska February 12, 1891 this man worked on a farm from 1906 to 1936. He was in a quartz mine from 1936 until November, 1942. On September 10, 1940 x-ray inspection revealed no evidence of silicosis (Fig. 8a) but on January 6, 1942 the trunk and hilar shadows appeared enlarged and slight nodulation was seen (Fig. 8b). On November 11, 1942 more nodulation appeared and a cavity was described in the left upper lung field (Fig. 8c). The sputum contained acid fast bacilli. The only complaint was dyspnea. Following bed rest until February, 1943 the cavity was not seen and sputum was negative for acid fast bacilli (Fig. 8d).

A silicotic individual may develop tuberculosis with cavitation and have symptoms that are confused with advanced silicosis. Cough may develop with increasing dyspnea and the individual finally collapses from fatigue before tuberculosis is discovered as illustrated by the following case:

Case 9, W.J.S.: On March 20, 1947 this patient gave a history of having worked as a quartz miner since February, 1933. On January 20, 1942 he was told he had advanced silicosis and advised to quit work. This he did in February, 1942. At this time he had shortness of breath on exertion but no cough. He then worked at an Arms Plant until January, 1944 and for the next seven months as a railroad brakeman. Following this he was a cab driver in Denver for eleven months and nine months in a war industry plant. In September, 1945 he began work in a Terra Cotta plant where analysis revealed about 35 per cent free silica in the air.

Sixteen samples of the dust collected showed from 5 to 5½ million dust particles per cubic foot at the breathing zone of the cutting space. Air in other parts of the room contained 4,000,000 per cubic foot. This, according to the standards given previously, is not adequate to produce silicosis. In September, 1946 this man began to cough, have chest pain and raise sputum. These symptoms grew worse and on November 11, 1946 tuberculosis with beginning cavitation was found in both lungs, in addition to silicosis. The sputum revealed acid fast bacilli. He discontinued work on December 5, 1946. X-ray inspection showed evidence of diffuse nodular infiltration throughout both lung fields consistent with silicosis and in the mid-portion of each lung evidence of cavitation was seen (Fig. 9a). On bed rest his cough decreased and he had no fever even

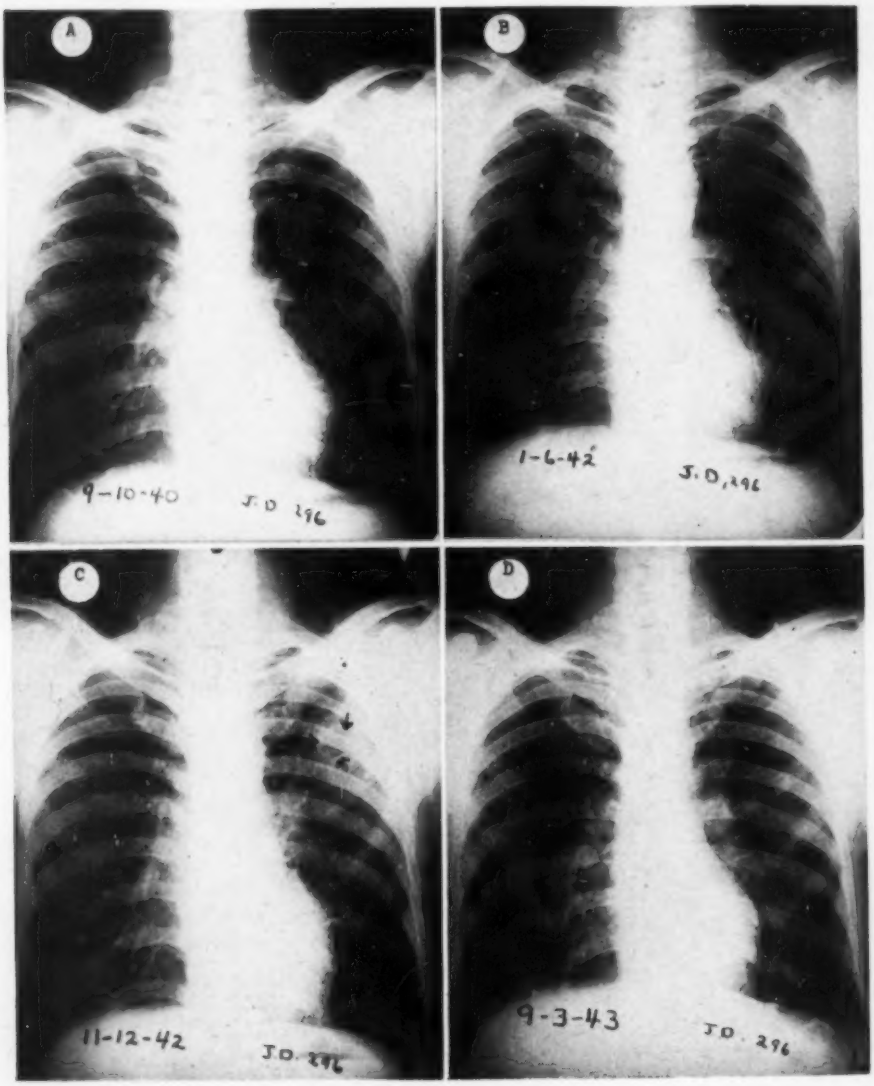

FIGURE 8, CASE 8:
Showing the development of tuberculosis in a silicotic with subsequent healing.

following a hemorrhage on March 6th. On March 24, 1947 the cavities appeared smaller (Fig. 9b) and the sputum contained less than 10 tubercle bacilli per field. He appeared well.

Miners with silicosis often do not complain of symptoms until some accident happens, as the following case and some of the previous ones illustrate:

Case 10, A.T.C.: When examined December 13, 1937, this man of 52 years stated that he had worked in quartz mines most of his life. He had a back injury March 21, 1933 for which he was granted compensation. At that time he complained of shortness of breath and exhaustion which he said were not present before the accident. Examination revealed infected tonsils, increased bronchial breathing throughout the chest with few asthmatic wheezes, slight enlargement of the liver, advanced silicosis and enlargement of the heart to the left. Superimposed tuberculosis could not be excluded (Fig. 10).

Prevention of Silicosis

As shown by Lanza,[18] prevention of silicosis is entirely possible if engineering methods can be applied to control or remove the dust at its source. This is expensive, but "so is the provision of safe drinking water. People have been educated to spend enormous sums of money to provide safe drinking water but they have not learned to be as intelligent about providing a safe atmosphere to work in." Medical supervision is as essential in the prevention of silicosis as the installation of engineering controls. In some places it is almost impossible to entirely control the escape of silica dust into the atmosphere. Industry which has a silica dust

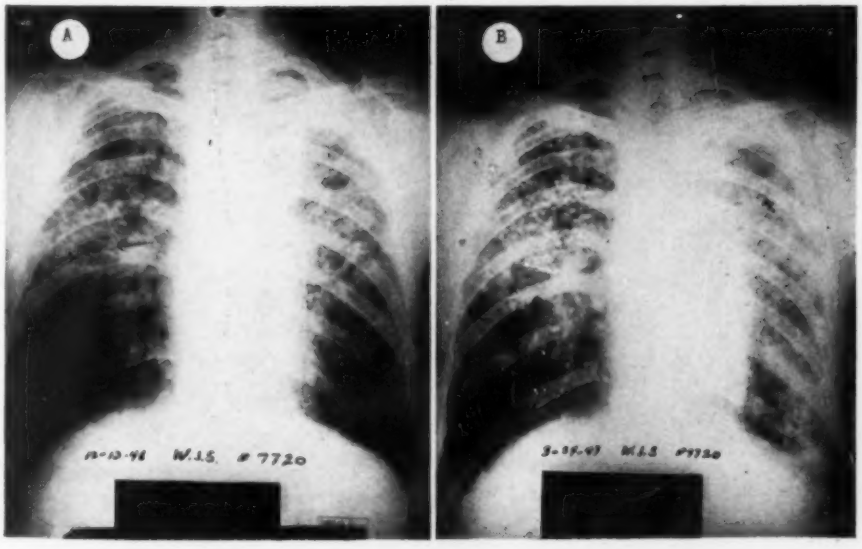

FIGURE 9, CASE 9:
Showing development of tuberculosis in a far advanced case of silicosis.

hazard should be under medical supervision. Although the actual supervision of safety measures must necessarily be the responsibility of competent foremen, experiences show that workmen quite often, despite medical and supervisional warning, will not take necessary precautions which are available to protect them from a dust hazard.

The medical department should see that workers have frequent chest x-ray inspection for evidence of early silicosis, and at the first sign of its development advise other employment. The physician should show the x-ray films to the worker and explain the condition. In this way Hamlin[19] found the employees expressed an appreciation of the interest taken in them and better followed the physician's advice. Many had refused to wear respirators but when informed they evidenced new interest in the protection of their health. Our suggestion is that x-ray be more generally used in pre-employment and at regular intervals thereafter.

Laws are now in effect or are being passed in all states to allow workmen's compensation for silicosis. They might be established to permit the employer to dismiss a worker at once, without interference from the union, if he does not carry out safety rules.

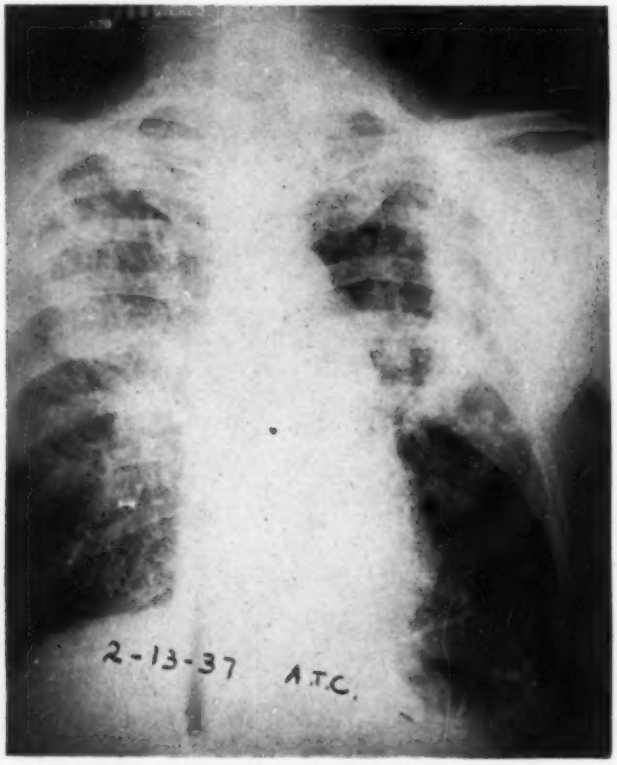

FIGURE 10, CASE 10

Unfortunately we lack cooperation. If only the labor unions would realize that we are all on the side of humanity the path would be easy.

Treatment

In 1936 Denny, Robison and Irwing[20] reported that inhalation of finely divided metalic aluminum powder would inhibit the action of silica in the lungs of experimental animals. Gardner[21] and his co-workers used hydrated alumina with good results both in inhibiting action of silica and the relief of symptoms in silicotic patients. There is some evidence, according to Gardner and others, that hydrated alumina may cause an increased susceptibility to tuberculosis, and should be used with caution. Bamberger[22] concluded that hydrated alumina seems superior to the metalic powder for the treatment of silicosis. Research work on humans has been carried on at the University of Colorado School of Medicine since October 1945, by Dr. J. W. Berry[23] on the treatment of silicosis with hydrated alumina. A detailed report on results will soon be published.

Aluminum hydrate is now being used by some companies as a direct inhalation or dispersed into the atmosphere of change houses as a prophylactic measure before the men enter underground mines. However, since silicosis takes years to develop, it will require considerable time to determine whether the inhalations of aluminum dust or hydrated alumina will prevent the development of this disease in man.

All agree that the use of aluminum should in no way interfere with the engineering control and medical supervision of the dust hazard in industry. It may prove a great adjunct to the prevention of silicosis where the escape of silica dust into the atmosphere is difficult to control.

SUMMARY

1) The history, terminology and classification of silicosis are given.

2) The role of the size of the silica particle, the number per cubic foot of air, the concentration of silica in a free state, the time of exposure, the humidity of the working atmosphere, and the condition of the ciliated epithelium, as conditions influencing the production of silicosis are discussed.

3) Differentiation from other chest diseases is emphasized.

4) Serial periodic x-ray studies of men exposed to silica dust, showing their progression from one stage to another are presented.

5) Cases are included showing: (a) The role of pre and post silicotic infection in the development of silicosis; (b) The peculiar

variability in the response of various workmen to silica dust during and after exposure; (c) That in some cases silicosis continues to develop even though the worker is removed from silica dust.

6) Engineering and medical controls in the treatment and prevention of silicosis are discussed.

RESUMEN

1) Se presenta la historia, terminología y clasificación de la silicosis.

2) Se discute el tamaño de la partícula de sílice, número de partículas por pie cúbico de aire, la concentración de la sílice en estado libre, el tiempo de exposición, la humedad de la atmósfera en que se trabaja y el estado del epitelio ciliado como condiciones que influyen la producción de silicosis.

3) Se recalca su diferenciación de otras enfermedades del pecho.

4) Se presentan estudios radiográficos seriados de hombres expuestos al polvo silíceo, que demuestran su progreso de una etapa a otra.

5) Se incluyen casos que demuestran: (a) el papel de la infección presilicótica y postsilicótica en la producción de la silicosis; (b) la variabilidad peculiar de la respuesta de diferentes trabajadores al polvo silíceo durante y después de la exposición; (c) que en algunos casos la silicosis continúa desarrollándose aunque se aparta al trabajador del polvo silíceo.

6) Se discuten los controles médicos y de ingeniería en el tratamiento y prevención de la silicosis.

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Primary Atypical Pneumonia: Roentgenographic Course, Complications, Recovery Rate, and End Results*

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Many recent communications to the medical literature on primary atypical pneumonia have emphasized the difficulty of differentiating that disease from other pneumonic consolidations from the roentgenogram alone.[1-6] It is generally recognized that the various pulmonary inflammations do, on x-ray examination, have a tendency to group types: there are so-called "typical" pictures for pneumococcal lobar consolidation, for broncho-pneumonia, or for chronic Friedlander's bacillus pneumonia. But the intergroup resemblance is so closely akin to the intragroup resemblance, with considerable overlap, that the usual inability or justifiable unwillingness of the roentgenologist to make an etiologic diagnosis of the acute pneumonic process on an x-ray film is immediately apparent.

It has been shown, for example, by Stein and Kresky[4] that in the early and resolving stages of pneumococcal pneumonia it is impossible to distinguish the infiltration from that of atypical pneumonia. Lobar consolidations of non-pneumococcal origin occur frequently, as do segmental or lobular consolidations of both bacterial or virus origin. Thus, it has been made increasingly clear that the nature of an acute pulmonary inflammation is a clinical problem. The various clinical studies, bacteriologic studies of the sputum, laboratory examinations of the blood, including cold isoagglutinin studies, and when indicated, special skin tests, should determine the etiology. The roentgenogram is then used to aid in determining the extent of the process, the efficacy of therapy, the rate of recovery, and the presence of complications and sequelae. Once determining that a "pneumonia" is present, the roentgenologist's task is almost purely a descriptive one. This, of course, excludes those cases of chronic pulmonary inflammations when the roentgenologist may be of inestimable aid in determining the ultimate diagnosis.

A term used frequently is "pneumonitis." This is employed by some as generally synonymous with "pneumonia," by some as synonymous with primary atypical pneumonia, and by many as

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a diminutive, usually meant to indicate a "small area of consolidation." Because of this multiplicity of usages, it is suggested that the term "pneumonia" be solely employed to denote any parenchymal inflammatory process of bacterial, virus, Rickettsial, or unknown origin, exclusive of the nontuberculous mycotic diseases, tuberculosis, and frank pulmonary abscess.

In some pulmonary diseases, notably tuberculosis, reasonably accurate predictions concerning the course of the disease, recovery rate and prognosis can be furnished by the roentgenologist on the basis of the roentgenographic characteristics of the lesion. The present study of 1857 cases of penumonia, occurring in a large Naval recruit training center, was made in an effort to determine whether, purely from a roentgenographic point of view, the distribution of involvement in primary atypical pneumonia of unknown etiology bears any relation to the course of the disease, the presence of complications, the rate of recovery, and the end results.

Method: From January 1, 1945 to July 31, 1946, 29,948 patients received diagnostic roentgenographic examinations by the x-ray Department. Of this number, 1857 had a roentgenographic diagnosis of "pneumonia." From this number were discarded all those cases clinically diagnosed as lobar (pneumococcal) pneumonia, bronchopneumonia complicating the exanthemata, postoperative pneumonia, and all pneumonias of determined or undetermined origin lacking adequate follow-up studies to the conclusion of the disease. This left a total of 855 analyzable cases of pneumonia clinically diagnosed as "primary atypical pneumonia of unknown etiology." These cases were all of males, ranging in age from 17 to 35. No attempt was made in this study to correlate the patient's age with the disease. No deaths occurred in this series.

As a routine procedure, all individuals having moderate or severe respiratory symptoms, with or without demonstrable abnormal physical signs in the chest, were examined by 14 x 17 chest roentgenograms. In this way many clinically unsuspected cases of "small patches of consolidation" were uncovered especially during the months of February-May 1945 and December 1945-March 1946, when the incidence of primary atypical pneumonia at this station reached epidemic proportions. Once a roentgenographic diagnosis of pneumonia was obtained, the patient was re-examined by x-ray at intervals of from 5 to 10 days (most usually every 7 days) until roentgenographic evidence of complete resolution was obtained. This was performed despite clinical evidence of recovery. No effort was made to correlate clinical evidence of recovery with radiographic recovery, but as a general observation, the former occurred earlier than the latter.

When the findings on the original roentgenogram were equivocal, re-examination was performed in 48 to 96 hours. If there were clinical evidence of complications, recurrences, reinfections or "spreads," then re-examinations including oblique, lateral, and stereoscopic projections were obtained as indicated. Fluoroscopic examinations were performed in selected cases.

Findings: According to the x-ray findings, these cases were placed into two main groups (Table 1): those showing unilobar involvement throughout the course of the disease, and those having multilobar involvement. The ratio of unilobar cases to multilobar cases was 4.4:1. Of the unilobar cases, the incidence of occurrence in the upper lobes and in the right middle lobe was almost equal, and by far, the majority of cases involved one of the lower lobes, with almost equal incidence in these cases. Of the multilobar

TABLE 1

DISTRIBUTION OF 855 CASES OF PNEUMONIA BY LOBE INVOLVED

	No. Cases	Per cent
1. *Unilobar Involvement* (including recurrences):		
A. Right upper lobe	41	4.9
B. Right middle lobe	48	5.6
C. Right lower lobe	256	29.9
D. Left upper lobe	59	6.9
E. Left lower lobe	272	31.8
Unilobar Total	676	79.1
2. *Multilobar Involvement* (including re-infections and "spreads"):		
A. Both lower lobes	116	13.5
B. Entire right lung	2	0.2
C. Entire left lung	4	0.5
D. All five lobes	4	0.5
E. Other multilobar combinations	53	6.2
Multilobar Total	179	20.9
TOTAL	855	100.0

TABLE 2

RECURRENCES

No. Cases	Lobe Involved	Average Time of Recurrence	Average Clearing Time
9	Right lower	14 days	22.0 days
1	Right middle	7 days	11.6 days
1	Left upper	6 days	17.1 days
7	Left lower	17 days	24.8 days

cases, involvement of both lower lobes predominated. Seventy-five and two tenths per cent of the 855 cases involved one or both lower lobes.

The entire right lung was involved only twice, and the entire left lung was involved 4 times. All five lobes were involved in 4 cases, and all other multilobar combinations occurred in 53 cases.

In 18 cases, an incidence of 2.1 per cent recurrences occurred. For the purpose of this study, a recurrence was defined as a reconsolidation of the same lobe or portion of the lobe during resolution, or re-consolidation after resolution but before complete subsidence of clinical signs or symptoms. A patient, once discharged as recovered, was treated as a new case if he developed pneumonia after radiographic and clinical recovery. Table 2 shows the number of cases, location, average time of recurrence after initial roentgenographic diagnosis of pneumonia was made, and the average total clearing time.

In 27 cases, or 3.1 per cent, reinfections or "spreads" occurred. A reinfection, or "spread" was defined as involvement of one or more originally uninvolved lobes during the disease, or after roentgenographic resolution but before subsidence of clinical signs or symptoms. In 13 of the 27 cases thus recorded (Table 3) "spread" occurred from one lower lobe to the other.

Complications occurred in 108 cases (Table 4). Pleurisy, as evidenced by parietal pleural thickening, interlobar septal thickening, and blunted costophrenic angles, but without evidence of free pleural or interlobar fluid, occurred 35 times, or in 4.1 per cent. Free pleural fluid was present in 58 cases, 53 of which were with ipsilateral pneumonia, 3 with contralateral pneumonia, and in 2 cases bilateral effusion occurred. In 12 cases unilateral effusion occurred with bilateral pneumonia.

When, in the course of a pneumonia, a contralateral effusion occurred, as in the above mentioned 3 cases, as well as in the 2 cases of bilateral effusion, there arose the question of "idiopathic" pleural effusion of tuberculous origin. All 5 such cases promptly

TABLE 3
RE-INFECTIONS OR "SPREADS"

No. Cases	Original Site	Re-infection or "Spread" Site	Average Time After Initial Onset	Average Clearing Time
9	right lower	left lower	7 days	20.2 days
4	left lower	right lower	10 days	25.1 days
14	all other combinations		12 days	23.4 days

recovered; the period of observation was limited only to the acute process, but it was reasoned clinically that the effusion was purely on the basis of the inflammatory process, and thus the patient was released to duty when clinically and roentgenologically recovered. Re-examination at a later date was usually suggested.

Interlobar effusion was present 4 times, once in the left septum, once in the right major septum, and twice in the minor septum. Loculated pleural fluid developed twice, in both cases on the right side.

Partial atelectasis occurred 5 times, an incidence of 0.5 per cent. Partial atelectasis of the right lower lobe occurred twice, and there was one case each involving the right upper, right middle, and left lower lobes. One case of complete atelectasis of the right lower lobe occurred.

Movitt[7] has reported two cases of spontaneous pneumothorax in adults with pneumonia. This complication, producing a 30 per cent collapse of the right lung occurred in one case of right lower lobe pneumonia. Pericardial effusion, observed clinically and roentgenographically, was present in one case. One case of spon-

TABLE 4

COMPLICATIONS

A. Pleurisy			35
B. Free pleural effusion			58
1. Ipsilateral		53	
a. With contralateral pneumonia	12		
b. Without contralateral pneumonia	41		
2. Contralateral		3	
3. Bilateral		2	
C. Interlobar effusion			4
1. Left		1	
2. Right major		1	
3. Right minor		2	
D. Loculated pleural fluid (right)			2
E. Atelectasis			6
1. Partial		5	
a. Right upper lobe	1		
b. Right middle lobe	1		
c. Right lower lobe	2		
d. Left lower lobe	1		
2. Complete, right lower lobe		1	
F. Others			3
1. Subcutaneous emphysema		1	
2. Pneumothorax, right		1	
3. Pericardial effusion		1	
COMPLICATIONS TOTAL			108

taneous subcutaneous emphysema occurred, and is reported in detail below.

Infiltrations of an acute pneumonic nature, especially in the upper portions of the lung fields, have frequently been confounded with active tuberculosis.[1,8] Lesions of primary atypical pneumonia frequently resemble the "typical" soft exudative reinfection apical or subapical tuberculous process. These are seen in serial films to resolve with the expected rapidity of a pneumonia, under proper therapy. Reinfection tuberculosis was thus suspected 35 times in this series. Twenty-one cases of right upper lobe pneumonia, and 14 cases of left upper lobe pneumonia were acid-fast suspects. None, after intensive clinical and laboratory studies, proved to be tuberculosis. All lesions resolved completely. No case of active tuberculosis was present in the entire series.

The end results are shown in Table 5. Of these cases 90.0 per cent had roentgenographically completely normal lung fields, without evidence of permanent or semi-permanent changes. Four and nine tenths per cent retained parietal pleural thickenings (as evidenced by viscero-parietal diaphragmatic "tentings," thickening of the axillary pleura, or blunted costophrenic sinuses). Interlobar pleural thickenings remained in 6 cases, and localized parenchymal fibrotic changes remained in 12. These latter categories should possibly have had further roentgenographic studies at a later date to determine whether such changes that were

TABLE 5
END RESULTS

Findings	No. Cases	Per cent
Completely normal lung fields	778	90.9
Parietal pleural thickenings	42	4.9
Left lower lobe pneumonia	23	
Right lower lobe pneumonia	17	
Interlobar pleural thickenings	6	0.7
Left upper lobe pneumonia	2	
Right middle lobe pneumonia	3	
Right lower lobe pneumonia	1	
Localized fibrotic changes	12	1.4
Right lower lobe pneumonia	5	
Left lower lobe pneumonia	7	
Suspected (unproved) bronchiectasis	13	1.6
Bronchiectasis	4	0.5

demonstrated were of a permanent or semi-permanent nature; such studies, however, were impracticable because of the brevity of the training period for these men, and were, in general, only suggested in the patient's medical record for the cognizance of his future medical officer.

Suspected bronchiectasis posed some unusual problems. Prolongation of the clearing time, especially in lower lobe pneumonias, coupled with clinical evidence of protracted respiratory infection usually aroused suspicion. Equivocal or deceiving findings in bronchography during or soon following a severe acute respiratory infection have led to the recognition of so-called "pseudo-bronchiectasis." Bronchography was thus not attempted until good and sufficient proof existed clinically that the disease had progressed into a subacute or chronic process, and roentgenographically that little or no progress toward resolution was taking place. Bronchography therefore was postponed until 30-60 days had elapsed after the onset of the illness. Four cases of bronchiectasis, an incidence of 0.5 per cent, were obtained. Thirteen others were suspected, but unproved, and after a prolonged hospitalization and convalescence they recovered; the recommendation that further studies at a later date be instituted, if indicated, was usually made (Table 6). The 4 cases of bronchiectasis represent the only cases not returned to their Naval duties as cured (Table 7). Re-examination at a later date was advised to determine the permanency of the bronchiectasis.[8,9]

Cases discharged as recovered numbered 851. Table 8 shows the analysis of these cases, with reference to man-days of roentgen-

TABLE 6
SUSPECTED BRONCHIECTASIS

Location	Complication	No. Cases	Man-Days Clearing	Average Clearing Time
Right lower lobe	None	4	252	63.0 days
Left lower lobe	None	3	131	43.7 days
Both lower lobes	None	2	97	48.5 days
Right middle lobe	None	1	49	49.0 days
Both lower lobes	Pleural effusion	1	55	55.0 days
Right lower lobe	Pleural effusion	1	48	48.0 days
Left lower lobe	Pleurisy	1	88	88.0 days
TOTAL		13	720	
AVERAGE CLEARING TIME				55.4 days

TABLE 7
BRONCHIECTASIS

Location	Complication	Duration Before Diagnosis Made	Type of Bronchiectasis
Right lower lobe	None	66 days	Cylindrical
Left lower lobe	None	69 days	Cylindrical
Left lower lobe	Pleural effusion	71 days	Cylindrical
Left lower lobe	Subcutaneous emphysema	68 days	Saccular

TABLE 8
CLEARING TIME ANALYSIS

Site	Cases of Uncomplicated Pneumonia	Man-Days of Clearing	Average Clearing Time (days)	Cases of Pneumonia and Pleurisy	Man-Days of Clearing	Average Clearing Time (days)
Right upper lobe	41	658	16.0	0	0	
Right middle lobe	46	612	13.3	0	0	
Right lower lobe	233	3523	15.1	12	203	16.9
Left upper lobe	53	678	12.8	1	12	12.0
Left lower lobe	240	3430	14.3	9	209	23.2
Both lower lobes	99	1690	17.1	5	91	18.2
Others	47	602	12.8	8	163	20.4
TOTAL	759	11193		35	678	
Average Clearing Time (days)			14.7			19.4

Table 8 (Continued)

Site	Cases of Pneumonia and Effusion	Man-Days of Clearing	Average Clearing Time (days)	Total Cases	Total Man-Days	Average Clearing Time (days)
Right upper lobe	0	0		41	658	16.0
Right middle lobe	2	36	18.0	48	648	13.5
Right lower lobe	10	154	15.4	255	3880	15.2
Left upper lobe	5	147	29.4	59	837	14.2
Left lower lobe	20	322	16.1	269	3961	14.8
Both lower lobes	12	249	20.7	116	2030	17.5
Others	8	190	23.8	63	955	15.2
TOTAL	57	1098		851	12969	
Average Clearing Time (days)			19.3			15.2

ographic clearing time, and the average clearing time. These cases were placed in three groups: those with uncomplicated pneumonia, those with pneumonia and pleurisy, and those with pneumonia and pleural effusion, all in relation to the lobar distribution of the pneumonic process. Of course, the actual duration of the pneumonia before roentgenography in any individual case could not be determined; similarly, since it obviously was impractical to obtain films at intervals more frequent than 5-10 days, the actual day of resolution could not be determined either. It was reasoned that these two incalculable factors would naturally balance each other, and thus they are neglected in this study. Clearing time by x-ray, from the time the lesion was first discovered, was found to take any time from 2 to 88 days. No significant difference in clearing time for uncomplicated pneumonias in relation to the lobe involved was found, except for bilateral lower lobe involvement which took an average of 2.4 days more to clear. Approximately 5 days longer on the average was required for resolution if pleurisy or effusion occurred. No significant over-all difference in average clearing time for all cases, with and without complications, in relation to the lobe involved, occurred. A general over-all average of 15.2 days was required for x-ray resolution for the entire series of 851.

CONCLUSIONS

The distribution of involvement of primary atypical pneumonia apparently bears no startling statistical relation to the course of the disease, including the incidence of recurrence and reinfection, and thus there is little predictability in any individual case. Seventy-five and two tenths per cent of the cases involved one or both lower lobes. The general over-all average roentgenographic recovery rate for 851 recovered cases was 15.2 days; for uncomplicated pneumonia 14.7 days; for pneumonia with pleurisy 19.4 days; for pneumonia with effusion 19.3 days. Pleurisy did not occur with right upper or middle lobe pneumonias, and only once with a left upper lobe pneumonia. Effusion did not occur with right upper lobe disease, and only twice with right middle lobe involvement (4.2 per cent), but with the same incidence as in right lower lobe pneumonia. Bronchiectasis was suspected 13 times (12 in lower lobe involvements) and proved 4 times, 3 in the left lower and 1 in the right lower lobe. Seven hundred and seventy-eight cases, 90.9 per cent, had completely normal roentgenograms on recovery; minor postinflammatory changes were present in 60 cases, and these were exclusively the result of lower lobe pneumonias. Bronchiectasis in 4 cases was confined to the lower lobes.

CASE REPORT

W. J. S., an 18 year old white male Naval recruit was admitted on April 25, 1946 complaining of malaise, coryza, dryness of the throat, and a cough of two weeks' duration. The cough had been treated symptomatically for one week but had become progressively worse and was associated with slight dyspnea on effort. No chest pain was present and he had experienced no recent chest trauma. The cough was only slightly productive of non-bloody non-frothy mucoid sputum. A 35 mm. photofluorographic chest film in March 1946 on enlistment, was negative. The past history was non-contributory .Physical examination revealed a temperature of 101 degrees F., pulse rate of 110, respiratory rate of 22. There was no cyanosis. Subcutaneous crepitus was demonstrated on both sides of the neck, shoulders, and upper thorax anteriorly and posteriorly. Moderate injection of the pharynx was present. Diminished breath sounds, slight increase in tactile fremitus, and scattered moist rales were present in the left chest posteriorly below the scapula and in the axilla. The heart was normal, except for tachycardia. The remainder of the physical examination was not remarkable. The findings were considered inconsistent with a spontaneous pneumothorax and consistent with an atypical pneumonia. The red blood cell count was 4,206,000 per cmm., with 13.0 gm. hemoglobin. The white blood cell counts ranged between 10,500 and 12,700 per cmm., with a normal differential count. The urinalysis was normal.

Chest x-ray examination on admission revealed a flocculent increased density at the left base and behind the left heart border. No evidence of pneumothorax was present. No fracture of the rib cage was visualized. The heart and mediastinal structures were normal in size and contour. A fairly marked subcutaneous emphysema of the soft tissue planes of the neck, supraclavicular regions, and the upper portions of each side of the thorax was present.

It was assumed that a spontaneous pneumothorax had occurred during the period of ambulatory treatment, and that it had absorbed before the initial roentgenogram was obtained. It was reasoned further that the point of the pneumothorax occurred where the visceral and parietal pleura were both densely adherent to the chest wall in order for air to escape into the tissue planes. The subcutaneous emphysema disappeared in 5 days and the patient became afebrile in 4 days. Partial resolution of the pneumonic process occurred until May 31, 1946, 36 days later. Thereafter, no further clearing of any significance occurred. The patient began to experience paroxysms of violent coughing which gradually became productive of mucoid and mucopurulent sputum. The sputum was never copious; no acid-fast bacilli were found on repeated examinations. On the 57th day, bronchiectasis was entertained as a roentgenographic diagnosis. Repeated short febrile episodes thereafter delayed bronchography until the 68th day. Instillation of iodized oil into the bronchial tree revealed saccular bronchiectasis of the axillary and posterior branches of the left lower lobe bronchus.

SUMMARY

This study is a statistical analysis of 855 cases of primary atypical pneumonia of unknown etiology. It was undertaken to determine whether, from a roentgenographic point of view, reasonably

accurate predictions can be made concerning the course of the disease, the recovery rate and the end result, from the original location of the pneumonia. The location of the pneumonia by lobe involvement was correlated with complications, such as pleurisy or effusion, reinfection, recurrence, clearing time, and condition of the lungs at the conclusion of the disease.

Seventy-five and two tenths per cent of the cases involved one or both lower lobes, with almost equal incidence in the other 3 lobes, including multilobar involvements. The overall roentgenographic clearing time was 15.2 days with no correlation between lobe involved and recovery rate in uncomplicated cases. An average of 4 days more was required for clearing if pleurisy or effusion were complications. The presence of pleurisy or effusion bore no relation to the location of the pneumonia. Atelectasis, interlobar effusion, loculated effusion, spontaneous pneumothorax, pericardial effusion, and subcutaneous emphysema were rare complications.

Ninety and nine tenths per cent recovered with completely normal lung fields. Minor permanent or semi-permanent fibrotic changes such as parietal or interlobar pleural thickening and localized parenchymal fibrosis occurred in 7 per cent, due mainly to lower lobe pneumonias. Bronchiectasis was suspected in 13 cases because of prolongation of the clearing time, but bronchography did not confirm this impression. Four cases of lower lobe pneumonia developed true bronchiectasis. Care was exercised to postpone bronchography until the disease had progressed into a subacute or chronic stage, to exclude cases of "pseudobronchiectasis."

One case of primary atypical pneumonia complicated by the development of a spontaneous subcutaneous emphysema and saccular bronchiectasis is reported in detail.

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RESUMEN

Este estudio es un análisis estadístico de 855 casos de neumonía primaria atípica de etiología desconocida. Se llevó a cabo este estudio a fin de determinar si, desde el punto de vista roentgenológico, es posible hacer pronósticos razonablemente exactos acerca del curso de la enfermedad, el índice de reposición y el resultado final, basados en la ubicación original de la neumonía. Se puso en relación la ubicación de la neumonía por lóbulos invadidos con complicaciones, tales como pleuresía o derrame, reinfec-

ción, recidivas, tiempo de aclararse y el estado de los pulmones al fin de la enfermedad.

En el 75.2 por ciento de los casos estaban invadidos uno o los dos lóbulos inferiores, con una frecuencia más o menos igual en los otros 3 lóbulos, inclusive de invasiones multilobulares. El tiempo total de aclararse la roentgenografía fue de 15.2 días y, en casos no complicados, no hubo relación alguna entre el lóbulo invadido y el índice de reposición. Se necesitó un promedio de cuatro días más para aclararse en casos complicados por pleuresía o derrame. La presencia de pleuresía o derrame no tuvo ninguna relación con la ubicación de la neumonía. Complicaciones raras fueron atelectasia, derrame interlobular, derrame loculado, neumotórax espontáneo, derrame pericardíaco y enfisema subcutáneo.

El 90.9 por ciento recuperó con campos pulmonares completamente normales. En el 7 por ciento ocurrieron cambios fibrosos menores, permanentes o semi-permanentes, tales como engrosamiento pleural parietal o interlobular y fibrosis localizada del parénquima, debidos principalmente a neumonías de lóbulos inferiores. Se sospechó bronquiectasia en 13 casos debido a prolongación en el tiempo de aclararse, pero la broncografía no confirmó esta opinión. En cuatro casos de neumonía en lóbulos inferiores se desarrolló bronquiectasia verdadera. Se tomó cuidado de aplazar la broncografía hasta cuando la enfermedad hubo progresado a una etapa subaguda o crónica, a fin de excluir casos de "seudobronquiectasia."

Se informa en detalle sobre un caso de neumonía primaria atípica complicada por el desarrollo de enfisema subcutáneo espontáneo y bronquiectasia sacular.

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Functional Examination of Each Lung Before and After the Paralysis of the Phrenic Nerve*

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In a previous work we started the study of the influences of different methods of collapse-therapy—and particularly pneumothorax—on the respiratory function of each lung. We are presenting here the results of the air content analysis of each lung before and after the surgical paralysis of the phrenic nerve. Gianotti and Ceruti[2] have seen in tracheotomized dogs that the ventilation of the lungs and the oxygen consumption diminishes after the excision of the phrenic nerve and that these effects persist after several weeks. The reduction in the respiratory values is somewhat higher in the bilateral than in the unilateral paralysis. Brea and Ferrari[3] made a functional study of seven cases of pulmonary tuberculosis before the paralysis of the phrenic nerve and also a few days and again one month after the surgical paralysis (by excision, alcoholization or crushing). After a few days they found a diminution of the voluntary apnea, the vital capacity, the respiratory volume per minute, the oxygen consumption, CO_2 elimination, and in the O_2 content of the alveolar air. Due to the development of compensatory mechanisms all these changes return to normal values one month after the intervention. Cournand and Richards,[4] in eight cases of therapeutic phrenic paralysis found striking impairment of the pulmonary function, as evidenced by decreased breathing reserve, unsaturation of arterial blood and a distinct tendency to pulmonary congestion after intravenous infusion. According to Rossier, Petzold, Decker and Michaud, cited by Steinlin,[5] the paralysis of phrenic nerve results in an unsaturation of arterial blood during moderate work and sometimes even during rest. To explain this finding it is suggested that although there is a reduced ventilation, the amount of blood circulating through the lower part of the homolateral lung remains constant. A pendular phenomenon in the air of lungs due to the paradoxical movement of the diaphragm has also been described.

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Bronchospirometric examinations in patients with surgical paralysis of the phrenic nerve were efected by Jacobaeus[6] (1 case) and by Pinner, Leiner and Zavod[7] (3 cases). Jacobaeus observed marked reduction of the ventilation, of the O_2 consumption and the disminution of CO_2. We believe that a bronchial obstruction existed which annuled nearly completely the functional value of the corresponding lung. Pinner, Leiner and Zavod admit that the paralysis of the phrenic nerve may have a considerably disfavourable effect on the pulmonary function.

Technique

Functional examination of the separate lungs was performed in ten cases of pulmonary tuberculosis (seven males and three females) before and after the therapeutical paralysis of the phrenic nerve obtained by: excision (3 cases), alcoholization (3 cases), crushing (3 cases) and phrenicotomy (1 case). In seven cases phrenic paralysis was produced on the right side and in the three others on the left. Functional studies were performed a few days before the intervention and between one and two months afterwards, except in two cases which were studied four and five months later respectively. The specific timing of the examinations was selected in order to eliminate the influence of the favorable or unfavorable progress of tuberculous lesions on the results and in this way to record only the differences due to the phrenic paralysis. We did not perform the examination immediately after the operation because we wished to avoid the immediate desequilibrium usually following surgery, and to permit the motor, sensory, vascular and trophic changes due to the phrenic paralysis to develop. Furthermore, with the study of each lung we could avoid any error produced by compensation of the other lung. The aim of our study was the observation of the more permanent physiological changes which occur after phrenic paralysis.

The bronchiospyrometric examination was performed with the Gebauer's and Zavod's gauges using the technique described by Vaccarezza and colleagues.[8] With the patient in sitting position the following indices were determined: vital capacity, complementary air, reserve air, respiratory volume, oxygen consumption and equivalent of ventilation.

For the comparative study of the results before and after surgical intervention the relative values of the different indices were determined for the separate lungs. We did not use the absolute values because these may be altered in each determination by the emotional reaction of the patient or by the anaesthesia. Comparative values between lungs are valid because in each analysis both lungs were examined under the same conditions.

Results

The paralysis of the phrenic nerve produced in all cases (see Table I), a reduction of the bronchiospyrometric indices of the corresponding lung with exception of the complementary and the reserve air, which in some cases even showed an increase.

The diminution in O_2 consumption was very marked (41.7 per cent). This means that the phrenic paralysis reduces almost to half the oxygen uptake of the blood in the corresponding lung.

The respiratory volume was diminished in all cases with the exception of case 7 in which a slight increase was observed. The mean of the total observations shows a reduction of 17.7 per cent.

The vital capacity was also reduced in all cases with the exception of number 6 in which almost no change was demonstrated, the mean being 14.9 per cent. The complementary and the reserve air had a mean diminution of 14.7 per cent and 19.6 per cent respectively. However considering that both indices are included in the vital capacity (and this never increases), the small increments found in such cases are only apparent. This fact can be easily explained if one recognizes that during the examination many patients, because of the anaesthesia or due to emotional factors, vary the degree of thoracic breathing from one determination to the next. This fact may, of course, affect the relative values of the components of the vital capacity, especially in relation to the complementary and reserve air.

TABLE I

Percentage of the Reduction in the Bronchiospyrometric Factors After Paralysis of the Phrenic Nerve

Observations	Vital Capacity	Complementary Air	Reserve Air	Respiratory Volume	Oxygen Consumption
1	.....	.....	.....	18.5	59.7
2	11.7	60.3	21.2	5.2	64.1
3	.....	.....	.....	22.2	60.2
4	10	+14.3	42.3	16.9	31
5	21.6	20.5	38.5	12.1	31
6	+ 2	+36.5	+16.2	60.5	52.9
7	7	34.6	8	+ 4.4	21
8	28.4	28.5	55.4	18.5	27.8
9	24.9	42.5	8.3	11.3	40.4
10	18.2	+17.4	42.3	16.4	29.7
Mean of the percentage of reduction	14.9	14.7	19.6	17.7	41.7

The equivalent of ventilation increased in all cases with the exception of case 6. This functional deterioration indicates the more marked reduction of the oxygen consumption in relation to the respiratory volume. The decrease in values of the different indices are as follows of: (1) oxygen consumption, (2) reserve air, (3) respiratory volume, (4) vital capacity and (5) complementary air.

Discussion

Changes observed in the different bronchiospyrometric indices show no strict relationship to the intensity of the diaphragmatic ascension. This is particularly manifested in cases 2, 3, 6 and 10.

One interesting fact is that the phrenic paralysis affects in a much higher degree the oxygen consumption than the other indices which are expressions of the function of ventilation. This predominating effect on the oxygen uptake of the blood can not then be explained on the basis of the unilateral paralysis of the diaphragm only.

It is known that the phrenic nerve has connections with the cervical sympathetic and the solar plexus and that it contains non-myelinated fibers. These two facts may indicate that this nerve has vasomotor effects on the pulmonary circulation, and this may explain why its section should produce important changes in the blood supply of the lung. It would be very interesting to determine the cause of this diminution in blood supply and the correlated diminution in oxygen consumption. To achieve this end, laborious experimentation which is beyond the scope of the present work would be necessary.

The differences observed between the two bronchiospyrometric examinations together with the short time elapsed between the phrenic paralysis and the second examination permit us to discard the presence of compensatory phenomena in the corresponding lung. In our studies of the separate functional characteristics of each lung such compensation was not observed. This compensation was however found by Brea and Ferrari[3] by examining both lungs together.

We do not want to extend our discussion to the clinical importance of these observations. However it seems very interesting to compare the results here observed with those found after the artificial pneumothorax. Paralysis of the phrenic nerve reduces to nearly half the functional value of the corresponding lung. Pneumothorax almost always improves the equivalent of ventilation of the collapsed lung due to a smaller reduction in oxygen consumption in relation to the respiratory volume.

SUMMARY

Separate functional examinations of each lung were performed in ten patients with pulmonary tuberculosis before and after the paralysis of the phrenic nerve.

In the lung on the paralyzed side the following percentage of diminution of bronchospyrometric values were observed: oxygen consumption 41.7; reserve air 19.6; respiratory volume 17.7; vital capacity 14.9; complementary air 14.7.

Due to the marked reduction in oxygen consumption in relation to the lesser reduction in respiratory volume the equivalent of the ventilation is frankly impaired.

The surgical paralysis of the phrenic nerve reduces almost to half the functional value of the corresponding lung.

RESUMEN

Se llevaron a cabo exámenes funcionales separados de cada pulmón en diez tuberculosos pulmonares, antes y después de la parálisis del nervio frénico.

En el pulmón del lado paralizado se observaron los siguientes porcentajes de disminución de los valores broncoespirométricos: consumo de oxígeno, 41.7; aire de reserva, 19.6; volumen respiratorio, 17.7; capacidad vital, 14.9; aire complementario, 14.7.

Debido a la decidida reducción en el consumo de oxígeno en relación a la menor reducción en el volumen respiratorio, el equivalente de la ventilación está francamente perjudicado.

La parálisis quirúrgica del nervio frénico reduce casi a la mitad el valor funcional del correspondiente pulmón.

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Photofluorographic Survey of 33,971 Apparently Healthy Persons in Greece

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INTRODUCTION

The present paper includes the first results of a survey carried out by the Photofluorographic Center established at the Rizarion School in Athens. This survey has been of very great importance for Greece, in view of the unreliability of the tuberculosis mortality statistics, and in view of the fact that the law of compulsory notification of infectious diseases does not apply to tuberculosis in Greece. The establishment of the Athens photofluorographic center at the end of 1945 can be considered to be a pioneering effort in public health in Greece. Since April 1946, a second dispensary has been operating in Salonika, both centers being under the direction of the Central Committee for investigation of the Incidence of Tuberculosis in Greece.

Up to the end of 1946, the Athens Center has examined 70,000 persons and the Salonika Center 20,000. In addition, a Mass X-Ray Survey Center established by the Tuberculosis Section of UNRRA during the summer of 1946 examined 10,000 persons in Patras. Surveys are carried out by examining apparently healthy groups, i.e. personnel of the Armed Forces, of Educational Institutions, of the Public Services, of mixed enterprises and factories and special groups. A 35 mm. miniature film is taken in the first place, and of those examinees whose films exhibit suspected pulmonary tuberculosis a large x-ray film is then taken, and the final diagnosis is made on the basis of the large film, combined with a clinical examination of the individual. Definite tuberculous examinees are then notified and informed about their condition, and receive pertinent advice.

The Athens Center conducts its own follow-up of suspects and of definite cases, because Greek State Dispensaries lack the means and the modern organization which are necessary for properly carrying out this service. Unfortunately, no laboratory facilities are available in the Athens Center for carrying out such tests as examination of sputum, blood sedimentation rates, etc. However, it is hoped that with the establishment of the Institute for Chest

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Diseases in Athens, which will incorporate the Photofluorographic Center, this need will soon be fulfilled.

Clinically significant cases are those of active pulmonary tuberculosis, the diagnosis of which is based upon the physical, x-ray, and laboratory findings. The clinically significant cases are subdivided into observation and treatment cases. Observation cases are limited to those which present no physical findings, and wherein the x-ray film demonstrates evidence of productive lesions tending towards fibrosis. The fact that each clinically significant case has been checked very carefully by our service gives special importance to our findings. The clinically insignificant cases consist of those whose x-ray film shows fibrotic or fibro-calcified lesions due to reinfection tuberculosis. Cases of healed primary lesions are not included among them.

This study includes the result of the mass survey of 33,971 persons belonging to apparently healthy groups of urban population. These groups are classified into six categories, according to their occupation.

The first category is composed of groups of the Armed Forces which were divided into three subgroups. The first subdivision consists of groups of candidates for the military schools, the second consists of cadets who successfully passed a physical examination

TABLE I

M A L E S

Ages	Examined	Clinically Significant Cases						Total of Clinically Significant Cases	Non-Clinically Significant Cases	Total Clinically & Non-Clinically Significant Cases
		Treatment			Observation					
		Min.	Mod. Adv.	Adv.	Min.	Mod. Adv.	Adv.			
15-19	2017	12	14	2	11	..	.	39	62	101
20-24	11173	79	78	7	76	6	1	247	352	599
25-29	4153	30	39	4	20	2	.	95	217	312
30-34	2736	40	42	4	25	8	.	199	187	306
35-39	2377	27	34	8	28	3	.	100	187	287
40-44	1839	30	28	3	26	3	1	91	177	268
45-49	1308	21	22	7	20	2	.	72	148	220
50-59	1362	27	42	10	18	8	1	106	169	275
60-69	458	7	16	2	3	1	.	29	61	...
70 & over	37	..	2	..	..	..	.	2	2	4
TOTAL	27460	273	317	47	227	33	3	900	1562	2462

TABLE II

FEMALES

Ages	Examined	Clinically Significant Cases						Total of Clinically Significant Cases	Non-Clinically Significant Cases	Total Clinically & Non-Clinically Significant Cases
		Treatment			Observation					
		Min.	Mod. Adv.	Adv.	Min.	Mod. Adv.	Adv.			
15-19	910	4	5	1	5	1	.	16	32	48
20-24	1900	14	18	4	15	4	.	55	79	134
25-29	1282	12	13	8	16	3	.	52	91	143
30-34	825	8	6	1	4	1	.	20	65	85
35-39	582	2	4	2	9	2	.	19	61	80
40-44	404	4	4	1	3	..	.	12	51	63
45-49	276	3	1	1	5	..	.	10	30	40
50-59	265	2	1	..	1	1	.	5	36	41
60-69	61	..	2	..	..	..	.	2	10	12
70 & over	6	..	..	..	..	..	.	...	2	2
TOTAL	6511	49	54	18	58	12	.	191	457	648

TABLE III

BOTH SEXES

Ages	Examined	Clinically Significant Cases						Total of Clinically Significant Cases	Non-Clinically Significant Cases	Total Clinically & Non-Clinically Significant Cases
		Treatment			Observation					
		Min.	Mod. Adv.	Adv.	Min.	Mod. Adv.	Adv.			
15-19	2927	16	19	3	16	1	.	55	94	149
20-24	13073	93	96	11	91	10	1	302	431	733
25-29	5435	42	52	12	36	5	.	147	308	455
30-34	3561	48	48	5	29	9	.	139	252	391
35-39	2959	29	38	10	37	5	.	119	248	367
40-44	2243	34	32	4	29	3	1	103	230	333
45-49	1583	24	23	8	25	2	.	82	178	260
50-59	1627	29	43	9	19	9	1	110	205	315
60-69	519	7	18	3	3	1	.	32	71	103
70 & over	43	..	2	..	..	..	.	2	4	6
TOTAL	33971	322	371	65	285	45	3	1091	2021	3112

shortly before they were x-rayed by our team. The third consists of the regular personnel of the Armed Forces.

The second category is composed of groups of students, the third category of personnel of the Public Services, the fourth category of personnel of "mixed enterprises" (i.e. Oil Companies, Telephone, Water supply, Electric Railways and Gas Companies). The fifth category is composed of factory groups and the sixth of special groups such as displaced persons, prisoners, registered prostitutes, etc.

The following tables, I to III, illustrate our statistical findings of the total of examinees, broken up into sex and age.

In analysing our findings, the following interesting facts come to light:

1) *Tuberculosis Incidence among the total of Examinees:*

The survey included 33,971 persons, 6,511 females and 27,460 males. Tables I, II and III show the result on the total of examinees according to sex and age. Of the entire number 1,091 were found to be clinically significant cases of pulmonary tuberculosis; 900 males and 191 females. Although the males constituted 80.9 per cent of the examinees they provided 82.4 per cent of the clinically significant cases.

In males, the incidence curve of tuberculosis increases with age whereas in females the highest rates are observed in age groups,

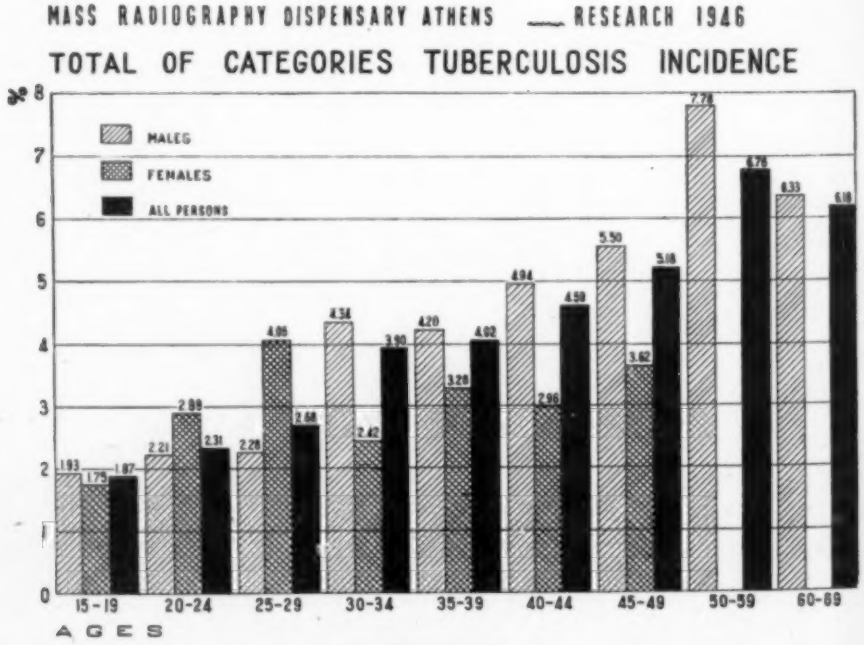

CHART I

20 to 30. Although the curve in the older age groups shows certain rises in the case of females, it is nevertheless always lower than the curve of incidence in males. The lower incidence among females in the age groups 35 to 50, compared with the higher curve for males is probably explained by the fact that females in these age groups are not engaged in occupations outside their homes.

Incidence figures for males (in age groups) similar to these discovered in Athens have been found through mass radiography surveys by Edwards in 1940 and by Elkin, Irwin and Kurzhalz in 1946, in the United States. In our survey, the average age of the examinees was 29.5 years and that of the clinically significant cases was 33.7 years.

2) *Tuberculosis Incidence among the different Categories:*

Differences in tuberculosis incidence were observed among the various categories. Chart II shows the incidence in our six categories from the highest to the lowest figures. Thus the category of "Armed Forces Personnel" with 13,582 examinees shows the lowest incidence, 209 clinically significant cases, i.e. 1.54 per cent. In the category of "Educational Institutions" with a total of 3,658 examinees, we find 124 clinically significant cases, i.e. 3.38 per cent. The "Public Service" category with a total of 6,382 examinees included 295 such cases, i.e. 4.62 per cent. The category of "Mixed Enterprises" with a total of 4,889 examined persons produced 243

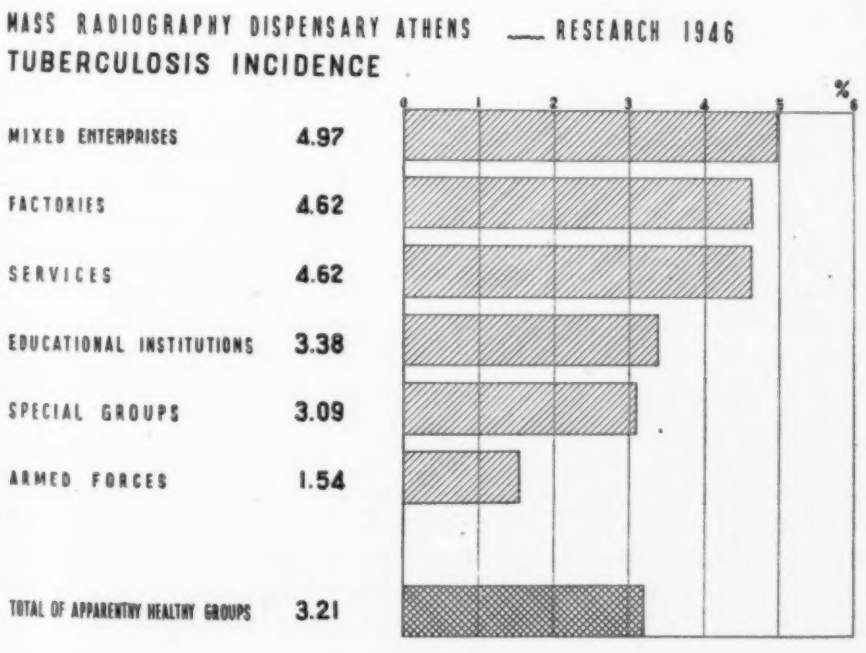

CHART II

cases, i.e. 4.97 per cent. The "Factories" category with 3,328 examinees provided 154 cases, i.e. 4.62 per cent. Finally, the sixth category containing the "Special groups" yielded 66 cases out of a total of 2,132 examinees, i.e. 3.09 per cent.

The comparatively low incidence found in the "Armed Forces" category (1.54 per cent) can be attributed to the strict physical examination to which the Army personnel are subjected at the time of recruitment, to the medical supervision received while in service and to the immediate dismissal from the Army of all detected cases of tuberculosis.

The relatively high mean incidence in the "Educational Institutions" category (3.38 per cent) is in keeping with the high incidence observed among the applicants for the University (3.28 per cent). One is greatly impressed by the high incidence found among the students of the Polytechnical School (5.10 per cent) which may be explained by the facts that: a) no medical examination is required of the students before their admission to the school, b) known tuberculosis cases are not removed from the school by the educational authorities and the disease is thus allowed to spread from the affected students to their fellows, c) the program of studies is exceedingly heavy.

Candidates for the University (3.28 per cent) and for the Cadet Schools (1.82 per cent) are in the same age group. The great differences in incidence rates between them may be explained by the fact that the cadets know that they must undergo a strict physical examination before being admitted to the Cadet Schools and therefore, possibly, apply a process of self elimination if they are cognizant of any disqualifying conditions obtaining among themselves.

In the "Public Services" category (4.62 per cent), the highest incidence was observed among employees of the Institute of Social Insurance (5.40 per cent). Bank of Greece employees also provided a relatively high incidence rate (4.24 per cent). In this latter group the highest incidence was found among personnel working in the underground offices of the Bank and at the counters where used bank notes are handled. The lowest incidence in this category was observed among the Greek employees of UNRRA (3.04 per cent).

In the category of "Mixed Enterprises" (4.97 per cent), the highest incidence was observed among the personnel of the Shell Oil Company (7.19 per cent). This incidence is in keeping with their high tuberculosis mortality rate (nine deaths from tuberculosis within two years, from the liberation in October 1944). The lowest incidence in this category was found among the personnel of the Gas Company (2.80 per cent).

In the "Factories" category (4.62 per cent), the highest incidence was observed in the Calyx Ammunition factory group (6.57 per cent) and the lowest among the personnel of the Eriourgia Textile Factory (3.91 per cent).

The striking variations in incidence observed between various groups within the 3rd, 4th and 5th categories are related to the kind of occupations, the medical supervision obtaining in these groups and, in the case of some enterprises, to the immediate dismissal of known cases of tuberculosis.

The comparatively low mean incidence in the sixth category containing the special groups (3.09 per cent) was greatly influenced by the low rates found among registered prostitutes.

The highest rate was observed among displaced persons (13.67 per cent). Unfortunately, our survey of Displaced Persons only includes a small number of examinees because when our team began to operate, most of the Displaced Persons had returned to their homes. Nevertheless, although the number of examinees was small, we considered the data reliable in view of the results recorded in other countries which were similar, when relatively large numbers were surveyed.

Comparing our figures with those obtained among similar groups in England and the United States, it is obvious that higher incidence rates are to be found among all groups in Greece. Thus, the percentage in Public Servants and Factory groups in Greece is between 4 and 7 per cent, whereas the percentage discovered in a survey carried out by the British Medical Research Council among similar groups was between 1 and 1.5 per cent. According to a Philadelphia survey, carried out by Elkin, Irwin and Kurzhalz in 1942, of 71,767 civilians working in war factories, the percentage was 2.3. In a survey, conducted by the United States Public Health Service in 1944, of 875,904 individuals belonging to factory groups, the percentage was 1.3.

Likewise, incidence rates among candidates for the Armed Forces of Greece (1.82 per cent) are higher than in the United States, where, for example, Schiller, in a survey of 40,273 applicants for service in the Armed Forces (in Buffalo) discovered an incidence rate of 0.45 per cent.

3) *Extent of the Disease:*

Of the total of 1,091 clinically significant cases, 607 (55.6 per cent) were minimal, 416 (38.1 per cent) moderately advanced and 68 (6.3 per cent) far advanced. There were 758 cases requiring treatment (68.5 per cent) and 333 observation (31.5 per cent).

Of the treatment cases, 42.48 per cent were minimal, 48.94 per cent moderately advanced and 8.58 per cent far advanced while

of the observation cases, 85.59 per cent were minimal, 13.51 per cent moderately advanced and 0.9 per cent far advanced. In the survey of the Factory groups by the United States Public Health Service, 67.7 per cent were minimal, 26.9 per cent moderately advanced and 5.4 per cent far advanced. Out of 1,091 clinically significant cases, 732 were newly discovered cases (67.1 per cent). Chart III shows the distribution of newly discovered and of known cases according to the extent of the disease and broken down into treatment and observation cases. It must be pointed out that of the newly discovered cases, 45.07 per cent were moderately advanced and 9.06 per cent were far advanced.

4) *Contribution to the Epidemiological Survey:*

The great number of newly discovered cases (67.1 per cent) shows how important is the contribution to public health offered by our survey.

In a large number of clinically significant cases diagnosed as minimal, restoration to health was obtained after a short period of treatment and the patients were able to return to their occupations. This is yet another proof of the great importance of mass radiography in early diagnosis of tuberculosis. Even in advanced countries where notification of disease is compulsory and an efficient public health service is available, a certain number of cases of tuberculosis fail to be registered. Moreover, a great number of

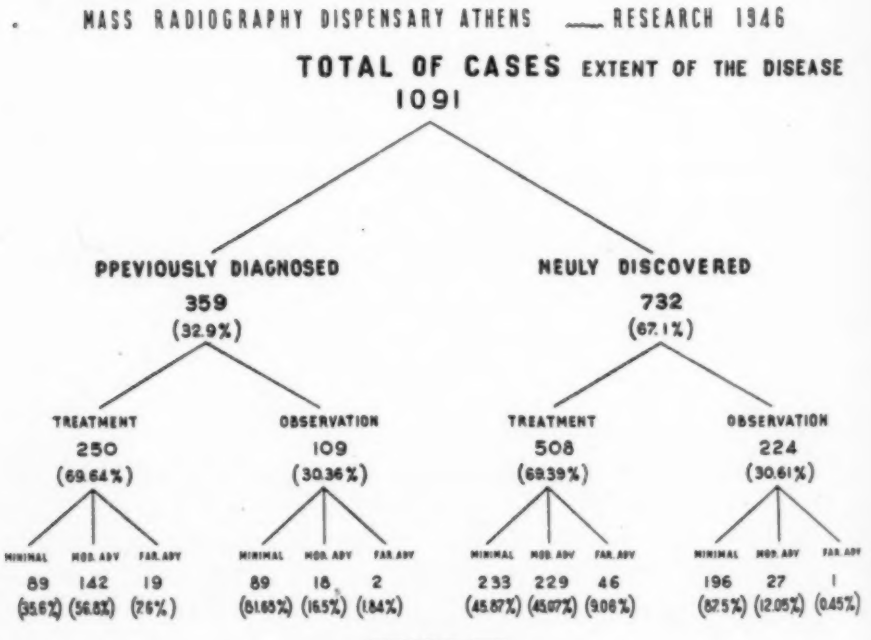

CHART III

suspected early cases can only be reached by mass radiography.

Our investigations so far have been carried out among urban groups only and therefore, our conclusions can only apply to the urban population. We hope, however, that by using mobile mass radiography units, we shall very soon be able to extend our survey to the rural areas of Greece.

The tuberculosis incidence in our survey was 3.28 per cent in males, 2.93 per cent in females and 3.21 per cent for both sexes. If our sex composition of our examinees were similar to that of the population (i.e., an equal number of both sexes with a small excess of females), the percentage for both sexes would be reduced to 3.11 per cent. It can be added that similar figures were obtained through surveys carried out in the towns of Salonika and Patras. The average age of our examinees was 29½ years, and therefore lower than the average age of the population. If we estimate the incidence of our examinees aged 29½, based on data obtained for both sexes in this age group (males 2.68 per cent and females 4.05 per cent), and if we adjust the sex ratio to that of the population, we obtain the incidence rate of 3.36 per cent.

It is difficult to determine the average age of our population after the war and the occupation. It was affected before the war by a high infantile and pre-school age mortality. During the occupation, owing to malnutrition and, at a certain period, famine, a great number of individuals in the older age groups died of starvation. At the present time, according to estimates drawn up by the Ministry of Reconstruction, the average age of our population is slightly lower than 40 years. In case the mean age of our group (29½ years) were correspondingly higher (about 40 years), the average incidence rate found (3.21 per cent) would certainly also be higher. Taking all the above into consideration, we think that we have a good foundation for estimating the incidence rate of tuberculosis among the urban population at about 3.5 per cent.

SUMMARY

A mass x-ray examination of 33,971 individuals, belonging to groups of Armed Forces, Educational Institutions, Public Services, Mixed Enterprises, Factories and Special groups gave the following results:

1) Of the total of our examinees, 1091 persons (3.21 per cent) were found to be suffering from clinically significant tuberculosis. The tuberculosis incidence rate in males was 3,28 per cent and in females 2.93 per cent.

2) Of the total of 1091 clinically significant cases, 758 required treatment (68.5 per cent) and 333 observation (31.5 per cent), 607

were minimal (55.6 per cent), 416 moderately advanced (38.1 per cent) and 68 (6.3 per cent) far advanced.

Of the treatment cases 42.48 per cent were minimal, 48.94 per cent moderately advanced and 8.58 per cent far advanced; while of the observation cases 85.59 per cent were minimal, 13.51 per cent moderately advanced, and 0.9 per cent far advanced. Out of 1091 clinically significant cases, 732 were newly discovered cases (67.1 per cent).

3) The striking variations in incidence observed among various groups are due to various reasons such as: occupation, or lack of it; kind of occupation; difference in age among examinees; previous medical examination; medical follow-up and relief or absence of it among the personnel of enterprises, services and factories; and in some cases to the immediate dismissal of known cases of tuberculosis.

4) Comparing our figures with those obtained among similar groups in England and in the United States, it is obvious that higher incidence rates are found among all groups in Greece.

5) Differences are observed in the incidence curve of males and females. Thus the incidence curve in males increased with age, whereas in females the highest rates are observed in age groups from 20 to 30. Although the curve of the older age groups shows certain rises in the case of females, it is nevertheless always lower than the curve incidence in males.

6) On the basis of the statistical findings of our survey, the incidence rate of tuberculosis among the urban population is estimated to be about 3.5 per cent.

RESUMEN

Un examen radiográfico colectivo de 33,971 individuos, pertenecientes a grupos de las Fuerzas Armadas, Instituciones Educativas, Servicios Públicos, Empresas Mixtas, Fábricas y Grupos Especiales, dio los siguientes resultados:

1) Del total de las personas examinadas, se descubrió que 1091 (el 3.21 por ciento) sufrían de tuberculosis clínicamente significativa. La frecuencia de la tuberculosis en los hombres fue del 3.28 por ciento y en las mujeres del 2.93 por ciento.

2) Del total de 1091 casos clínicamente significativos, 758 requerían tratamiento (el 68.5 por ciento) y 333 observación (el 31.5 por ciento); 607 eran mínimos (el 55.6 por ciento), 416 moderadamente avanzados (el 38.1 por ciento) y 68 (el 6.3 por ciento) muy avanzados.

De los casos que requerían tratamiento el 42.48 por ciento eran mínimos, el 48.94 por ciento moderadamente avanzados y el 8.58 por ciento muy avanzados; mientras que de los casos que sólo

necesitaban observación el 85.59 por ciento eran mínimos, el 13.51 por ciento moderadamente avanzados y el 0.9 por ciento muy avanzados. De los 1091 casos clínicamente significativos, 732 fueron casos descubiertos por la primera vez (el 67.1 por ciento).

3) Las conspícuas variaciones en frecuencia observadas entre los varios grupos se deben a varias razones, tales como: ocupación, o falta de ocupación; clase de ocupación; diferencias de edad entre los examinados; previos exámenes médicos; investigaciones médicas consecutivas y auxilio o falta de auxilio entre el personal de empresa, servicios y fábricas; y, en algunos casos, a la inmediata despedida de los casos conocidos de tuberculosis.

4) Cuando se comparan nuestros datos con los que se han obtenido entre grupos semejantes en Inglaterra y en los Estados Unidos, es obvio que se encuentra una frecuencia más alta entre todos los grupos de Grecia.

5) Se observan diferencias entre las curvas de frecuencia de hombres y mujeres. La curva de frecuencia en los hombres asciende con la edad, mientras que entre las mujeres las frecuencias más altas se observan en los grupos etarios de 20 a 30. Aunque la curva en los grupos de edades más avanzadas muestra ciertos ascensos en el caso de las mujeres, siempre es más baja, sin embargo, que la curva de frecuencia entre los hombres.

6) Basada en los hallazgos estadísticos de nuestro censo, se calcula que la frecuencia de la tuberculosis entre la población urbana es de un 3.5 por ciento.

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CORRECTION IN THE MAY-JUNE ISSUE

In the article on "Tumors of the Anterior Mediastinum" by Milton S. Lloyd, M.D., F.C.C.P., published in the May-June issue of "Diseases of the Chest," on page 413 credit is given to Merck and Company as contributor of the drug (parachlorophenol). This statement is in error and is hereby corrected to read "I am indebted to Mallinckrodt Chemical Company, New York City, who contributed a generous supply of the drug."

PRESIDENT'S ADDRESS

Problems Persisting Through the Evolution of Medicine

MAJOR GENERAL SHELLEY U. MARIETTA (Retired)*
Washington, D. C.

There is much of interest and of value in the writings concerning the large field of the history of medicine, and ideas gleaned from such works are helpful in the proper evaluation of modern day procedure. As Winston Churchill once remarked in an address to the Royal College of Physicians, "the longer you can look back the further you can look forward."

Many of the busiest and most progressive practitioners of modern medicine have been so aware of the benefits derived from a study of the evolution of medicine that they have not only been students in that field but have, for the edification of those of us who are less gifted, written extensively on the subject. Osler (1913); McKenzie (1927); Haggard (1933); Castiglioni (1940); and Guthrie (1946) are but a few of the more recent authors to whom we are indebted.

The period of ancient medicine may be considered as beginning with the earliest attempts at healing of which there is any record and ending with the sixth century A. D. Primitive medicine in one or another form was in existence many centuries before the earliest records in Egypt, some 5000 B. C. Soon after the advent of life on this planet crude attempts must have been employed by heads of families or groups, chieftains, magicians, priests or others supposedly better qualified than the common herd to make decisions and to act. In general it may be said that from 5000 B. C. to 600 A. D. the physician had progressed from the status of a medicine man dependent upon dances, masks, incantations and other means of casting out the evil spirits; through the stages of experimentation with various medicaments and procedures, gaining a little knowledge and recording it, separating medicine from magic and religion; to the point of establishing schools, hospitals and libraries, regulating medical practice and especially to more intelligently observe cause and effect. Materia medica and pharmacy were established, if in somewhat elementary form; sanitation was, in Rome at least, understood and practised to a very considerable degree; dissection of the human body was permitted in some countries and anatomy and pathology were taking their essential places in enriching the field of medicine; there was acceptance, at least on the part of the doctor, of the fact that diseases were due to factors other than the action of spirits, the consequence of sin or the wrath of God. Previously many of the faith had felt, as Christian Scientists still do, that reliance upon medical procedure implies a lack of complete faith in religion. Haggard gives credit to Hippocrates for bringing about the reform in the temple cult of healing. His "accomplishment was to relieve the gods of their responsibility for the prevention and the treatment of disease and to place that responsibility where it belonged—squarely upon the shoulders

*Presented at the 14th Annual Meeting, American College of Chest Physicians, June 19, 1948, Chicago, Illinois.

of man." Care of the sick at public expense, the doctor drawing an annual salary based upon a municipal tax, was provided in 200 B. C. Specialization was already a factor to be reckoned with, causing Herodotus to remark about 450 B. C. that "the practice of medicine is so divided up amongst them that each physician is a healer of one disease and no more. All the country is full of physicians, some of the eye, some of the teeth, some of what pertains to the belly, and some of the hidden disease." It will be noted that there is considerable similarity to some of the problems of our days and the solutions are not yet clear.

Medieval medicine may be said to have extended from 600 A. D. to 1600 A. D. During this period there was a general regression of scientific medicine but the work was carried on with a lessened vigor through three channels: The South Italian School; Byzantine Medicine; and Arabian Medicine.

The strongest remaining offshoot and greatest factor in the perpetuity of medical science of that time was Arabian medicine. Following their conquest of half of the then known world the Arabs seized with appreciation and avidity upon the scientific knowledge of the conquered areas and applied themselves to master this field. Their accomplishments were rapid and so complete that in about one century they had surpassed their instructors, the conquered scholars, although they continued a more or less friendly competition with them.

In this period must be mentioned Roger Bacon (1214 A. D.) who was one of the first to indicate the necessity for original research instead of the blind acceptance of some then accepted authority, past or present.

During the medieval period of medicine it is apparent that, although the accomplishment as to progress was greatly diminished in volume, and that there was an actual degradation of medical practice in the greater part of the civilized world, there were certain recompenses when the picture as a whole is considered. The medicine of Hippocrates and Galen was kept alive and even added to somewhat in southern Italy and in Arabia. The Church, although for centuries it had subordinated normal medical procedure to the use of various religious forms or rituals, translated and preserved in it's archives a considerable medical literature extant at that time; further, by it's action in the sixteenth century to accord to lay medicine the freedom and the right to reinstitute it's domain in the field of medicine by the use of any and all reasonable means, the Church did inestimable service in the advancement of medical progress. Rulers lessened restrictions and many avenues previously barred to study were again made available.

Also there was a new enthusiasm for learning, for a new way of life, for progress and thus the stage was set for the Renaissance and for the development of modern medicine which began about the end of the sixteenth century.

The tremendous, amazing strides made in the past three hundred and fifth to four hundred years, and more especially in the past fifty years in medical science and in it's practical application to the problems of preserving health and treating disease, constitute one of the finest chapters in world history, one in which we, as members of the medical profession, may take just pride.

The period of modern medicine is so replete with important accomplishments that any attempt, no matter how concise, to even mention it's features within the limits of this discussion would be futile.

Since the time of I-em-hotep, an Egyptian who flourished about

4900 B. C. in the Nile valley and who was the first fairly authentic medical practitioner of note, certain vexatious and recurring problems have annoyed the medical profession. Among the most important of these have been: Control by Church or State; absorption of it's function by public services; medical teaching both pre and post graduate; research activities; and specialization.

The experience of the past amply demonstrates that the profession of medicine does not function best under the dominance of either Church or State and especially when the two are synonymous. Recall if you please that the period from 410 A. D. to 1550 A. D., when medicine was disdained and indeed learning as such was discountenanced, was concurrent with the so-called Dark Ages and the dominance of the Christian Church in all fields. Although this subject has been discussed at length in the literature it is perhaps as well not to belabor it but only to draw attention to it's history. Even in these enlightened days we occasionally encounter cases in which religious inclinations of one type or other conflict to some extent with our concept of good medical judgment. The ruling power or State has always regulated medicine rather more than less and on the whole for the good of the majority.

On occasions pernicious measures have been recommended and sometimes placed upon the statutes. Generally such regulations have been discontinued after experience has proved their unsuitability. The Hammurabic Code, carved on a diorite block and set up in Babylon in 2000 B. C., consisted of a body of civil and religious laws, many of which related to the medical profession, and was for the purpose of informing the populace of their rights. The medical caste in Babylonia was at this time highly organized with practice regulated in detail, scale of fees laid down and penalties provided for malpractice. Some penalties were severe as for example the loss of the hands in case of death of a patient following treatment for a severe wound or post operative loss of an eye. Certainly the penalties were more severe than present day laws provide but so were those for other offenses, this being characteristic of the times.

Well organized public medical service was set up in the Greek cities as early as the latter part of the sixth century B. C. Still earlier, hygiene in Egypt was highly developed and cleanliness of the dwellings, cities and even of person was regulated by law. In Rome, during the first century A. D., teachers of medicine were provided at public expense. There were both army and navy surgeons and physicians were appointed to care for the poor. These were well paid from public funds and were distributed in proportion to the population. In some localities they were elected to office for a given period and a certain experience and renown were necessary to gain such an office. It is evident then that some government control of the profession has been existent for at least the past 2500 years and that it is less rigid now than earlier. Rules and regulations seem to have always been for the same purposes and indeed there is a considerable similarity as to the general principles involved.

It is well within the memory of many of us here when the instruction in medical schools was largely didactic. Speakers became red faced in their oratorical efforts to expound to the students what the latter might better have read at another time from text books or journals. Theory versus practice. We find in ancient medicine a similar and long enduring situation, finally settled after centuries in favor of bedside teaching. Hippocrates the great observer and practical bedside teacher; Galen,

bombastic, theoretical, who loved words and arguments and who left for the Arabian successors to Greek medicine his methods, which they long followed. Eventually the Hippocratic principles returned with the Renaissance but the tide has always ebbed and flowed. Osler in one of his lectures had this to say: "Live in the ward. Do not waste the hours of daylight in listening to that which you may read by night. But when you have seen, read. And when you can, read the original description of the masters who, with crude methods of study, saw so clearly." A French writer said: "Formerly we generalized with few facts and many ideas; now we generalize with many facts and few ideas." This remark suggests the picture of a medical neophyte who, with the help of an incomplete history and an inadequate physical examination plus a considerable number of laboratory slips, some pertinent to the case at hand and some unessential, sits down to arrive at a diagnosis—many facts and few ideas.

The need for investigation, which could have at that time been another name for research, was appreciated centuries before Christ. In the beginning of medicine everything was necessarily based upon the trial and error method—empiricism—and it was not until about the seventeenth century that more accurate procedure was made possible by improved facilities, both in equipment and method. However Pythagoras in the fifth century B. C. is said to have taught study of causes as a means of gaining knowledge. Galen, in the second century A. D. made experimental studies which led to important discoveries in the physiology of the nervous system. Since the seventeenth cetnury there has been a markedly accelerated impetus in this field until it has become one of the largest and most important branches of medicine.

Research connotes "a critical and exhaustive investigation or experimentation having for it's aim the revision of accepted conclusions, in the light of newly discovered facts." (Websters Dictionary). While it is true that vitally important discoveries have occasionally been made accidentally, generally as an incident in the progress of some perhaps unrelated investigation, true research requires a first and perhaps the most important step of deciding what particular problem is to be studied. This infers that the choice is made by those thoroughly familiar with the general field of the study and with what other investigators are presently doing in that field. As is usually the case with the medical profession the pendulum swings frequently too far; research now seems high on the upswing. Many studies are undertaken ill advisedly either because of poor selection, lack of funds to carry through, lack of necessary equipment, overlapping or even covering of studies already under way or recently completed, or because competent workers in that particular field are not selected. Thus funds are dissipated, issues are confused and erroneous results are reported. We should also appreciate that compilation of statistics, gathering of data or theorizing as to possible implications of certain hypotheses, do not constitute research.

Of the many branches of medicine, probably only embryology and anatomy have been well covered by the studies done heretofore. Histology and pathology are well up on the list but possible developments with carbon isotopes and the improved microscope may bring further light into these fields. Of the many others but a few will be mentioned. Geriatrics should interest several of us here as being included in a rapidly growing section of the population. I look with some misgivings into the not distant time when half of the people of the United States

will be in the older age group, fifty or more years. This can not have a wholesome influence on the welfare of the nation, economically, culturally or strategically. Still it is the policy and the ethics of the profession to prolong life to the final gasp and preventive medicine is just getting well into it's stride. Perhaps in a few decades thinking will be in terms of euthanesia which is said to have been carried out so practically by the cave dwellers. When old persons became too feeble to climb the pole to the cave entrance they were left to the mercy of the wild beasts, and the others were freed from the burden of their care. There was no expense, only labor was involved at that time and no institutions or hospital beds had to be considered.

Physiology and physiological chemistry may be expected to benefit materially through the use of radioisotopes but these agents, because of the dangers inherent in their makeup, will be available only to individuals and institutions authorized, because of their training and facilities, to properly make use of them. Radiophosphorus in polycythemia vera and leukaemia; radioiodine in thyroid disorders, radiogold in malignancies of the lymphoid system and radiosodium in leukaemia, all have promise.

Synthetic chemical products constitute an intriguing field but here again research is limited to large institutions with extensive facilities and a permanently operating trained group of specialists for consultant and technical purposes. It is quite possible that astonishing new products may be made available to the medical profession later as has been done in the past. In this connection, recognition should be accorded certain of the large pharmaceutical firms which maintain extensive laboratories and labor constantly to produce refined and standardized products for the control or cure of disease. Theirs is an altruistic attitude which is highly commendable, as too much success with new discoveries could adversely affect the sale of some of their earlier products, thus outmoded.

The development of antibiotics has the research group agog. Penicillin has been quite well evaluated. Streptomycin soon will be. We are learning again that no remedy can ever be unequivocally good. Undoubtedly there will be more moulds offered for therapeutic purposes. The newest one of promise is chloromycetin which is particularly effective against some of the virus infections. Here is a sphere in which our own group may well find an opportunity to participate.

What of specialization, just now a subject of wide discussion. Specialists are mentioned in Egyptian medical records. Darius of Persia had specialists at his court about 530 B. C. Herodotus, about 450 B. C., remarked with apparently not full approval as to the number of medical specialists then in being. In the fifth century A. D. Susruta, a prominent physician of India, is quoted by Guthrie as saying of specialists: "He who knows only one branch of his art is like a bird with one wing." The fact that there have been specialists from the time of Egyptian medicine and that their relative proportion in the profession is increasing proves the public sponsorship of the arrangement. Approximately 50 to 60 per cent of the doctors in the United States at the present time are devoting all or a considerable proportion of their practice to some special field.

There are two reasons for this: First, physicians because either of a less arduous life, greater financial returns, supposedly increased prestige or sheer inability to keep abreast of the current in the constantly widening field of medicine, have been and will continue to limit their practice

to that segment which seems to promise them most. Whether or not this is a desirable situation or whether we approve of it, is beside the point. It exists. Second, patients in seeking the best in medical attention apply logically to some one whom they feel to be especially well qualified to give an opinion or to carry out the particular medical procedure. Equally logically, the choice goes to the doctor who has had more experience in the condition for which treatment is desired, ergo the doctor who limits his practice to this and like diseases, the specialist. All this may seem unnecessarily elementary but it provides simple answers to the frequent and querulous complaints that too many doctors are limiting their practice. So far as I know there are no means of controlling this tendency. The incompetent doctor, specialist or not, may very properly be penalized for malpractice when his performance falls below the commonly accepted standards but he can not be otherwise chastened. The one effective method, as I see it, to control the poorly qualified specialist, and at the same time protect the public which experiences considerable difficulty in obtaining reliable information on the matter, is the procedure known as certification. To the long established and well qualified physician in any field, whose renown is a matter of record, certification, or lack of it, may mean little. To the newcomer as a specialist, certification, with all that it implies in the way of professional recognition, listing in the medical associations and ethical publicity, may be the stepping stone to success.

To acquire certification the applicant must meet the requirements set up by competent medical authority and it is evident that the higher the requirements the better the standing, both professionally and with the public, of that specialty and the greater the prestige of it's membership. I am of the opinion that a competent specialist must have had a good grounding in medicine and must maintain a working knowledge of the parent science. It is equally apparent to me that an internist is not in position to give patients the best in the way of early and accurate diagnosis and treatment in all of the several spheres into which modern medicine has been divided by it's practitioners, without consultation with at least some of the specialists in these spheres.

Patients, rich as well as poor, tend more and more to visit medical schools, hospitals and clinics where they are offered the opportunity of being seen by various specialists and may be given laboratory tests as indicated, by trained technicians under the supervision of specialists, and incidentally at considerably less cost. They, as well as doctors, recognize the advantages inherent in such a system.

With this glimpse into the past of medicine we may the better reconcile ourselves with our present problems, and realize that we are considering not entirely new factors but often old ones in a new setting. The latter setting is of our own manufacture and is no more immutable than those of the past. The utmost care, consideration, and discretion are required as we modify it, and modify it we must, to meet ever new demands.

RICHARD H. OVERHOLT, M.D., F.C.C.P.

PRESIDENT

American College of Chest Physicians

1948-1949

Richard H. Overholt, M.D., F.C.C.P.
Installed as College President

Dr. Richard H. Overholt was installed as President of the American College of Chest Physicians at the Fourteenth Annual Meeting of the College in Chicago, June 17-20, 1948.

Dr. Overholt was born in Ashland, Nebraska. He received his A.B. degree from the State Teachers College, Peru, Nebraska, and he graduated from the University of Nebraska School of Medicine. He served a two-year internship at the University of Pennsylvania Hospital in Philadelphia and during the next three years was a Fellow in Surgery under the late Professor George Muller at the University of Pennsylvania Hospital. His interest in thoracic disease was kindled in Pennsylvania while he was working on the services of Dr. Chevalier Jackson, Dr. David A. Cooper, and Dr. George Muller.

Following the completion of his postgraduate surgical training in Philadelphia, Dr. Overholt went to Boston, Massachusetts, to join the Surgical Staff of the Lahey Clinic where he engaged in the practice of general and thoracic surgery for a period of eight years. He resigned from the Lahey Clinic to enter the private practice of thoracic surgery and became affiliated with the Tufts College Medical School where he became Clinical Professor of Surgery.

His principal hospital connections are the following: The Pratt Diagnostic Hospital, the New England Hospital, and the Cambridge Tuberculosis Sanatorium. He is consultant Thoracic Surgeon to the New Hampshire State Sanatorium, the Rhode Island State Sanatorium, and four county sanatoria in Massachusetts.

FOURTEENTH ANNUAL MEETING

American College of Chest Physicians

The College has concluded another successful meeting. The registration, as indicated below, was the largest on record for a College meeting, exceeding our last annual meeting by 130 physicians. There was a total of 785 physicians registered for the scientific assembly of the College and for the Session on Diseases of the Chest in the Scientific Assembly of the American Medical Association. They came from 43 states, the District of Columbia, Alaska, Hawaii, Puerto Rico, the Virgin Islands, and from 22 other countries. Over 200 physicians' wives and guests also attended the meetings. For the first time in the history of the College, a social program was planned for the ladies under the chairmanship of Mrs. Kenneth G. Bulley, Aurora, Illinois, wife of the President of the Illinois Chapter of the College. These activities were so successful that the officials of the College have agreed to plan similar activities for future meetings. The total attendance of physicians and their wives at the College meeting this year was well over one thousand.

The various committees charged with the arrangements for the annual meeting handled their assignments efficiently and effectively. It was one of the best conducted meetings ever presented by the College and the officials in charge of the arrangements are to be congratulated for this splendid achievement.

Registration

State	*Number Registered*
Alabama	4
Arizona	14
Arkansas	1
California	40
Colorado	21
Connecticut	4
Delaware	1
District of Columbia	12
Florida	14
Georgia	9
Idaho	1
Illinois	124
Indiana	17
Iowa	5
Kansas	5
Kentucky	12
Louisiana	4
Maine	2
Maryland	9
Massachusetts	16
Michigan	32
Minnesota	23
Mississippi	4
Missouri	34
Montana	1
Nebraska	5
New Jersey	22

State	*Number Registered*
New Hampshire	1
New Mexico	2
New York	63
North Carolina	10
Ohio	41
Oklahoma	8
Oregon	7
Pennsylvania	38
Rhode Island	3
South Carolina	6
Tennessee	12
Texas	27
Utah	1
Virginia	13
Washington	2
West Virginia	6
Wisconsin	27
TOTAL	703

U. S. Possessions	*Number Registered*
Alaska	1
Hawaii	3
Puerto Rico	2
Virgin Islands	1
TOTAL	710

Other Countries	*Number Registered*
Argentina	8
Australia	4
Brazil	2
Canada	23
Chile	1
China	3
Cuba	5
Ecuador	2
England	1
France	2
Guatemala	2
India	1
Iraq	1
Ireland	1
Korea	1
Mexico	10
New Zealand	1
Pakistan	1
Peru	2
South Africa	1
Spain	2
Venezuela	1
TOTAL	75
GRAND TOTAL	785

Dr. Charles M. Hendricks Receives Second College Award

Dr. Charles M. Hendricks, El Paso, Texas, Past-President of the College, was presented with the College Medal and Certificate of Award for meritorious achievement in the specialty of diseases of the chest at the annual meeting of the College in Chicago. The Award was made by Dr. Edward W. Hayes, Monrovia, California, Chairman of the Committee on College Awards, at the Annual Presidents' Banquet held at the Congress Hotel on June 19th.

Examinations for Fellowship

On the first day of the College meeting, 52 candidates were given oral and written examinations for Fellowship in the College. This is the second largest group to take the examinations.

Presidents' Banquet

Dr. Fred M. F. Meixner, Peoria, Illinois, was the Toastmaster for the Annual Presidents' Banquet and introduced the guests of honor. A Certificate of Merit was presented to Major General S. U. Marietta, the retiring president, in recognition of his valuable services to the College. Dr. Chevalier L. Jackson, Philadelphia, Pennsylvania, Chairman of the Council on Pan American Affairs of the College, introduced the guests of honor from all of the other countries, and the banquet closed with the awarding of the College Medal to Dr. Charles M. Hendricks, El Paso, Texas. Preceding the banquet, a cocktail party was given by the Illinois Chapter of the College.

Scientific Program

An outstanding scientific program was presented in Chicago under the Chairmanship of Dr. Richard H. Overholt. A new feature of the program this year was the presentation of a series of round table luncheon meetings. The demand for tickets for these informal luncheon meetings far exceeded the seating capacity of the rooms. It has been recommended that the Scientific Program Committee for the next annual meeting of the College plan a similar program of luncheon meetings. Members of the College should secure their reservations for these luncheon meetings as far in advance of the meeting as possible. The Committee on Arrangements for the Scientific Assembly, under the Chairmanship of Dr. Paul H. Holinger, is to be complimented for the excellent manner in which the scientific assembly was conducted.

International Luncheon

The Council on International Affairs of the College sponsored a luncheon meeting at which time the officials of the College from the other countries presented reports of College activities in their respective countries, and a number of scientific papers were presented on various aspects of chest diseases. Dr. Chevalier L. Jackson, Chairman of the Council on Pan American Affairs presided at this meeting and was assisted by Dr. Andrew L. Banyai, Milwaukee, Wisconsin, Chairman of the Council on European Affairs, and Dr. Harry C. Warren, San Francisco, California, Chairman of the Council on Pan Pacific Affairs.

Clinics

A series of interesting clinics was presented at various hospitals in Chicago and all of the clinics were well attended. The arrangements for these clinics were under the Chairmanship of Dr. Abel Froman, Chicago, Illinois and the transportation arrangements were under the Chairmanship of Dr. Johann Bornstein. The physicians attending the clinics were provided with transportation from the hotel to the hospitals and return.

Luncheon Meetings

Several important luncheon meetings were sponsored by various councils and committees of the College and interesting programs concerned with the activities of these councils and committees were presented by eminent authorities.

Conference of College Chapter Officials

The Annual Conference of College Chapter Officials was held at the Congress Hotel, Chicago, at a luncheon meeting on June 17 and Dr. Carl C. Aven, Atlanta, Georgia, Chairman of the Conference, presided. A program dealing with the activities of College chapters was presented by various members of the Conference. Dr. Charles A. Thomas, Tucson, Arizona, was elected Chairman of the Conference for the ensuing year and Dr. Irving Willner, Newark, New Jersey, was elected Secretary of the Conference.

New College Officers

Dr. Richard H. Overholt, Brookline, Massachusetts, was inducted into office as President of the College, and the Committee on Nominations presented the following slate of officers who were elected unanimously:

Joseph C. Placak, M.D., Cleveland, Ohio, President-Elect
Louis Mark, M.D., Columbus, Ohio, 1st Vice-President
Harry C. Warren, M.D., San Francisco, Calif., 2nd Vice-President
Minas Joannides, M.D., Chicago, Illinois, Treasurer

REGENTS

Regional District		*Term Expires*
Number 3	Martin H. Collier, M.D., Grenloch, New Jersey	1951
Number 7	James H. Stygall, M.D., Indianapolis, Indiana	1951
Number 9	Paul A. Turner, M.D., Louisville, Kentucky	1951
Number 11	Carl H. Gellenthien, M.D., Valmora, New Mexico	1951
Number 12	Robert B. Homan, Jr., M.D., El Paso, Texas	1951
Puerto Rico	Luis A. Passalacqua, M.D., Santurce	1949
Peru	Ovidio Garcia Rosell, M.D., Lima	1949
South Africa	David P. Marais, M.D., Cape Town	1949
Venezuela	Jose Ignacio Baldo, M.D., Caracas	1949

All present Regents in other countries were re-elected.

GOVERNORS

State		*Term Expires*
Alabama	William S. Armour, M.D., Birmingham	1951
California	Buford H. Wardrip, M.D., San Jose	1951
Connecticut	Cole B. Gibson, M.D., Meriden	1951
Georgia	Carl C. Aven, M.D., Atlanta	1951
Illinois	Robert K. Campbell, M.D., Springfield	1951
Louisiana	Alton Ochsner, M.D., New Orleans	1951
Massachusetts	Hubert A. Boyle, M.D., New Bedford	1951
Minnesota	Karl H. Pfuetze, M.D., Cannon Falls	1951
Mississippi	Robert E. Schwartz, M.D., Hattiesburg	1951
Missouri	Alfred Goldman, M.D., St. Louis	1951
New Jersey	Irving Willner, M.D., Newark	1951

State		*Term Expires*
New York	George Foster Herben, M.D., Yonkers	1951
Tennessee	David H. Waterman, M.D., Knoxville	1951
Utah	William R. Rumel, M.D., Salt Lake City	1951
Vermont	Albert G. Mackay, M.D., Burlington	1951
Wyoming	Carleton O. Anton, M.D., Sheridan	1951

Governors in U. S. Possessions

Alaska	Anibal R. Valle, M.D., Seward	1951
Hawaii	William F. Leslie, M.D., Hilo	1951
Puerto Rico	A. M. Marchand, M.D., Santurce	1949

Governors in Other Countries

Australia New South Wales	William Cotter Harvey, M.D., Sydney	1949
Peru	Juan A. Werner Ortega, M.D., Lima	1949
Venezuela	Julio Criollo Rivas, M.D., Caracas	1949

All present Governors in other countries were re-elected.

Report of the Treasurer

FINANCIAL STATEMENT, MAY 31, 1948

1 CASH ASSETS				
Checking Account, First National Bank of Chicago			$35,211.78	
Special Funds:				
Life Membership Bonds, Series G		$ 1,300.00		
Life Membership Checking Account		2,085.00		
		$3,385.00		
Endowment Fund Bonds	$10,000.00			
Checking Account	603.42	10,603.42		
Research Council Checking Account		11,589.43		
General Reserve Fund, Bonds		10,000.00		
Total Special Funds			35,577.85	
Total Cash Assets				$70,789.63
2 BUDGET				
Budget for year ending December 31, 1948			$61,059.98	
Budget for last eight months, 1948		$44,500.00		
Checking Account balance		35,211.78		
Anticipated deficit		$ 9,288.22		
Budget for fiscal year ending December 31, 1949			65,700.00	
Increase in budget				$ 4,640.02
3 ANTICIPATED INCOME from all sources in 1949				$51,000.00

The increase in the budget for 1949 of $4,640.02 has become necessary because of the increase in prices of all items. Because of this inevitable increase in expenses and the necessity to meet financial responsibilities, it has been recommended to the Board of Regents of the College that ways and means be found to increase the College income.

Respectfully submitted, Minas Joannides, M.D., F.C.C.P.

Report of the Executive Secretary

Another year has come and gone since we met in Atlantic City and I am happy to report to you that it has been a year of progress.

Perhaps the most outstanding achievement during the past year has been the unanimous vote of the House of Delegates of the American Medical Association to establish a Section on Diseases of the Chest in their Scientific Assembly. While this Section is an integral part of the American Medical Association, yet this important achievement would not have been accomplished without the active support of the College. It now becomes an important duty of every College member to support this newly established Section by registering for the Section at the meetings of the American Medical Association and by attending the scientific sessions.

The establishment of this Section on Diseases of the Chest in the American Medical Association has stimulated the College chapters in urging the establishment of similar sections in their state medical societies. I am also happy to report to you that during the past year a Section on Diseases of the Chest has been established in the Scientific Assembly of the Medical Society of the State of New Jersey and in the Medical Society of the State of New York. The College officials in these states who helped to bring this about are to be congratulated. Movements are now in progress to establish similar sections in some of the other state medical societies and I am certain that the efforts of the College officials in those states will also be crowned with success. However, it is not enough to create these sections in the American Medical Association and in the state medical societies; they must be fully supported by the College membership if they are to serve a useful purpose.

Another outstanding achievement of your College during the past year has been the active program of your Council on Postgraduate Medical Education. The Second Annual Postgraduate Course in Diseases of the Chest was presented in Chicago under the auspices of this Council in September, 1947. Sixty-two physicians from 20 states, the District of Columbia, Canada, China, Cuba, Mexico and Spain were enrolled in this course. The first postgraduate course in diseases of the chest to be presented by the College in cooperation with the Laennec Society of Philadelphia was presented in that city in March of this year. Sixty-three applicants from 17 states, the District of Columbia, Canada and India were enrolled in this course. The registration for these courses had to be restricted because of the limited seating capacity and many more applications for both courses were received than could be accommodated.

The Chairmen and all of the members of the Council who directed these courses gave liberally of their time and efforts. Letters received from the physicians who participated in the postgraduate courses gave much praise to these officials, as well as to the instructors and administrators of the courses.

With this excellent achievement to its record, your Council on Postgraduate Medical Education has planned three additional courses in diseases of the chest to be presented in the fall of this year. A postgraduate course will be given in San Francisco from September 13 through September 17 in collaboration with the University of California Medical School and Stanford University School of Medicine. The Third Annual Postgraduate Course will be given in Chicago from September 20 through September 25, and the first postgraduate course to be given in New York City will be held from November 8 through November 12. Complete information on these postgraduate courses will be mailed to the members of the College as soon as it is available.

The companion to the Council on Postgraduate Medical Education is the Council on Undergraduate Medical Education. Your Council on Undergraduate Medical Education has conducted a constructive and energetic program over a period of years and their activities have culminated in the publication of two essential books for undergraduate medical students. One of these books, which is now on the press, is entitled "The Fundamentals of Pulmonary Tuberculosis and Its Complications (for the Student, the Teacher and the Practicing Physician)." The other book, now being compiled, will deal with non-tuberculous diseases of the chest. These books are being published by Charles C. Thomas Publishers, Springfield, Illinois. Those members of the College who are engaged in teaching will no doubt want to have the books made available to their undergraduate medical students, and we are certain that all members of the College will want to have copies of these excellent books in their libraries.

Another important part of our medical educational program is the presentation of scientific papers at the various international, national and sectional meetings of the College. To help make this possible, College chapters have been organized in practically every state and district in the United States and its possessions, as well as in many countries outside of the United States. These chapters, which in many cases meet with other organized medical societies, present an excellent forum for discussion of the latest developments in the specialty of diseases of the chest.

All of these College activities, which form the basis of our medical educational program, find expression in the official journal of the College, "Diseases of the Chest."

We have added pages to the journal to take care of the accumulation of scientific material. Circulation of the journal is now at its highest peak. We are happy to report that "Diseases of the Chest" is read by physicians in 53 countries. This excellent record is due to the efforts of your Editorial Board, under the able direction of its chairman, Dr. Jay Arthur Myers. Today "Diseases of the Chest" is recognized throughout the world as the outstanding publication in this specialty. This is an achievement of which all of the members of the College may be justly proud.

Notices of the College activities during the past year have been given prominence in the various state and county medical society publications, as well as in the Journal of the American Medical Association. Numerous scientific journals in other countries have also carried accounts of College affairs. We are particularly grateful to the fine publicity given to the College activities in the "Revista Panamericana de Medicina y Cirugia del Torax." This journal, under the direction of

our able Regent for Mexico, Dr. Donato G. Alarcon, publishes the College news concerned with Latin American affairs, in the Spanish language for the benefit of our Spanish-speaking members. I would also like to point out that the scientific articles in this journal are published in both Spanish and English.

The present College By-Laws were adopted in June, 1942. During the past six years your College has more than doubled in membership and its activities have increased many times. It has accordingly become necessary to revise our present By-Laws and a committee to study the revision of the College By-Laws is now diligently engaged with this important task. The revised By-Laws will be submitted to the College membership as soon as they are available.

The international program of the College deserves special comment in the Report of your Executive Secretary. Our membership extends into 48 countries and we are pleased to report that despite the unsettled conditions existing in Europe, your Council on European Affairs has admitted 88 members in the following countries: Austria, Belgium, Czechoslovakia, Egypt, England, France, Greece, Hungary, Ireland, Italy, Lebanon, The Netherlands, Norway, Palestine, Portugal, Scotland, Spain, Sweden, Switzerland, Turkey and Yugoslavia.

The Council on Pan Pacific Affairs has also shown a marked increase in its membership, particularly in Australia, China and India. The most recent College chapter to be organized is the one for South Africa, where we have 21 members. As conditions become more stable, College chapters will be organized in many of the other countries.

The splendid achievement of our Council on Pan American Affairs is worthy of comment. With few exceptions, there are active College chapters in most of the Central and South American countries. The large delegations of our good friends from the countries to the north and to the south of the United States who attend our annual meetings, are evidence of the excellent accomplishments of our Council on Pan American Affairs.

College members in any part of the world can now travel to 47 other countries and find the welcoming hand of a Fellow of the American College of Chest Physicians. Truly, the American College of Chest Physicians is a world society.

No report of your Executive Secretary would be complete without an expression of appreciation for the fine cooperation received from your President and all of the other officials of the College. To the Assistant Executive Secretary, the College Auditor, and to all of the other members of the College staff, your Executive Secretary expresses his appreciation and thanks. Without their loyal support, this report of progress would not be possible.

We have come a long way in achieving many of the objectives of the American College of Chest Physicians. There is still much to be done. Our sights are set and we must not be deterred from reaching our goal. By perseverance and by diligently adhering to the principles which have been established for the College, we will continue to make progress.

Respectfully Submitted, Murray Kornfeld.

Annual Meeting, Board of Governors

The Board of Governors held their annual meeting at the Congress Hotel on June 17. The meeting was called to order at 10:00 a.m. by Dr. Robert K. Campbell, Springfield, Illinois, Chairman. The meeting was attended by the following Governors and alternates of the College:

State	
Arizona	Howell Randolph, M.D., Phoenix
California	Seymour M. Farber, M.D., San Francisco (alternate)
Colorado	Arnold Minnig, M.D., Denver
Delaware	Gerald Beatty, M.D., Wilmington
Florida	M. Jay Flipse, M.D., Miami
Georgia	Carl C. Aven, M.D., Atlanta
Illinois	Robert K. Campbell, M.D., Springfield*
Iowa	J. Carl Painter, M. D., Dubuque
Kentucky	T. Ashby Woodson, M.D., Louisville
Maine	Francis J. Welch, M.D., Portland
Massachusetts	Hubert A. Boyle, M.D., New Bedford
Michigan	Willard B. Howes, M.D., Detroit
Mississippi	Robert E. Schwartz, M.D., Hattiesburg
Missouri	W. W. Buckingham, M.D., Kansas City
New Jersey	Martin H. Collier, M.D., Grenloch
New York	George Foster Herben, M.D., Yonkers
Oklahoma	Robert M. Shepard, M.D., Tulsa
Oregon	James M. Odell, M.D., The Dalles
Rhode Island	Frank A. Merlino, M.D., Providence (alternate)
Texas	Charles J. Koerth, M.D., Kerrville (alternate)
Virginia	E. C. Harper, M.D., Richmond
West Virginia	George R. Maxwell, M.D., Morgantown
Wisconsin	Carl O. Schaefer, M.D., Racine
U. S. Army	Arden Freer, M.D., Washington, D. C.
Veterans Administration	Roy A. Wolford, M.D., Washington, D. C.
Canada (Ontario)	H. I. Kinsey, M.D., Toronto
Argentina	Manuel Albertal, M.D., Buenos Aires (alternate)
Cuba	Gustavo Aldereguia, M.D., Havana (alternate)
Ecuador	Jorge A. Higgins, M.D., Guayaquil
Venezuela	Jose Ignacio Baldo, M.D., Caracas

*Chairman, Board of Governors.

Dr. Joseph C. Placak, Cleveland, Ohio, Chairman of the Board of Regents of the College, presented an outline of the procedure for processing of applications for membership in the College. This was followed by a general discussion of the subject.

A resolution was introduced by the Governor of the College for California requesting that the duties of the Governor in the processing of applications for membership in the College be clarified in the College By-Laws. This resolution was adopted and referred to the Board of Regents.

Dr. Karl H. Pfuetze, Cannon Falls, Governor of the College for Minnesota, was elected by the Board of Governors as their representative on the Committee on Nominations.

Dr. Robert K. Campbell was re-elected as Chairman of the Board of Governors for the ensuing year.

Annual Meeting, Board of Regents

The Board of Regents of the College convened at the Congress Hotel, Chicago, Thursday, June 17 at 2:00 p. m. and again on Sunday, June 20 at 5:00 p. m. Dr. Joseph C. Placak, Cleveland, Ohio, Chairman of the Board, presided. The following Regents, alternates and invited guests attended the meeting:

Joseph C. Placak, M.D., Cleveland, Ohio, *Chairman*
Donato G. Alarcon, M.D., Mexico City, Mexico
Russell S. Anderson, M.D., Erie, Pennsylvania (invited guest)
Carl C. Aven, M.D., Atlanta, Georgia (alternate)
Andrew L. Banyai, M.D., Milwaukee, Wisconsin
Alvan L. Barach, M.D., New York, N. Y. (invited guest)
Otto L. Bettag, M.D., Pontiac, Illinois (invited guest)
L. J. Buis, M.D., Richmond, Virginia (alternate)
Robert K. Campbell, M.D., Springfield, Illinois
Frank S. Dolley, M.D., Los Angeles, California
Seymour M. Farber, M.D., San Francisco, California (invited guest)
Carl H. Gellenthien, M.D., Valmora, New Mexico
Edward A. Greco, M.D., Portland, Maine
E. C. Harper, M.D., Richmond, Virginia (invited guest)
Edward W. Hayes, M.D., Monrovia, California
Charles M. Hendricks, M.D., El Paso, Texas
Jorge A. Higgins, M.D., Guayaquil, Ecuador (alternate)
Robert B. Homan, Jr., M.D., El Paso, Texas
William A. Hudson, M.D., Detroit, Michigan
Chevalier L. Jackson, M.D., Philadelphia, Pennsylvania (invited guest)
Minas Joannides, M.D., Chicago, Illinois
Charles J. Koerth, M.D., Kerrville, Texas (invited guest)
J. George Lang, M.D., New York, N. Y. (invited guest)
Edwin R. Levine, M.D., Chicago, Illinois (invited guest)
S. U. Marietta, M.D., Washington, D. C.
Louis Mark, M.D., Columbus, Ohio
Donald R. McKay, M.D., Buffalo, New York (alternate)
Rene G. Mendoza, M.D., Havana, Cuba (alternate)
Jay Arthur Myers, M.D., Minneapolis, Minnesota
William E. Ogden, M.D., Toronto, Canada
Richard H. Overholt, M.D., Brookline, Massachusetts
J. Winthrop Peabody, M.D., Washington, D. C.
Sir Sidney Sewell, Melbourne, Australia
James H. Stygall, M.D., Indianapolis, Indiana
Harold G. Trimble, M.D., Oakland, California (invited guest)
Paul A. Turner, M.D., Louisville, Kentucky
William C. Voorsanger, M.D., San Francisco, California (invited guest)
Harry C. Warren, M.D., San Francisco, California

Murray Kornfeld, Chicago, Illinois, Executive Secretary
Harriet E. Lumm, Chicago, Illinois, Assistant Executive Secretary

Committee on Membership

Dr. Roy A. Wolford, Chairman of the Committee on Membership of the College presented the following report: As of June 1, 1948, there were 2578 members in the College, and 84 applications for membership were pending investigation. This shows an increase of 306 new members

admitted into the College during the past year. Of the 2578 members, 1860 are Fellows, 139 Associate Fellows, and 579 are Associate Members. In the United States of America and its possessions, there are 1886 members, while in countries outside of the United States, our membership totals 692. These are distributed over 47 countries. Since the report submitted last year by the Committee on Membership, members have been admitted from ten additional countries.

Committee on Certification

Dr. J. Winthrop Peabody, Chairman of the Committee on Certification, presented a report of the meeting of his committee with the representatives of the American Board of Internal Medicine at San Francisco in April of this year. The question of a certification board for diseases of the chest is now being studied by the American Board of Internal Medicine and the College Committee will submit a report to the Board of Regents upon receipt of further information from the American Board of Internal Medicine.

Editorial Board

Because of the accumulation of manuscripts accepted for publication in the College journal "Diseases of the Chest," the Editorial Board recommended that the journal be published monthly commencing with the January, 1949 issue. To help defray the cost of publication, the Editorial Board recommended to the Board of Regents that there be an increase in the annual dues of the College and that the subscription price of the journal also be increased. This resolution was approved by the Board of Regents and was referred to the Executive Council of the College for further study.

Council on Public Relations

Dr. William C. Voorsanger, Chairman of the Council, presented a report and that portion of the original report submitted to the Board of Regents at their meeting in Washington, D. C. in November, 1947, dealing with publicity and public relations as pertaining to medical journals and medical societies was adopted.

Council of Medical Directors and Superintendents of Tuberculosis Hospitals and Sanatoria

The following preliminary report of the Council was presented by Dr. Russell S. Anderson, Chairman.

This Council has now been functioning since approximately 1944. Dr. Benjamin L. Brock was its original chairman and resigned during the early spring of 1948 because he was no longer actively engaged in sanatorium practice, per se. The undersigned has, therefore, been functioning as chairman subsequent to that time.

The Council is composed of nine members under whom two committees are active with an additional combined membership of fourteen. One of these committees, of which Dr. Allan Hurst, Denver, Colorado, is chairman, has been assigned to the problem of devising recommendations in the field of rehabilitation. The other committee, whose chairman is Dr. I. D. Bobrowitz, Otisville, New York, is charged with a more complicated task; namely, the drawing up of a code for the standard-

ization of tuberculosis hospitals and sanatoria. These committees, and particularly their chairmen, have to their credit a prodigious amount of work on these respective projects.

Subsequent to the termination of the World War, the Council has sponsored a luncheon meeting during the annual convention of the College. The luncheon groups were planned to afford the hospital superintendent or medical director a forum wherein he can compare notes with his colleagues and air his problems peculiar to hospital management, both from the medical and administrative angles. The luncheon meeting held in Chicago, June 18, 1948, was well attended and the informal discussions were spontaneous and instructive. An additional hour could have been utilized with profit at this meeting.

A business meeting of the Council and its two committees was held Friday evening, June 18th. The following actions were taken by unanimous vote:

1) To request the Board of Regents to consider changing the name of the Council, for the sake of brevity, to "The Council of Tuberculosis Hospitals."
2) To accept the attached preliminary reports of the Committee on Rehabilitation and the Committee on Tuberculosis Hospital Standards and submit them to the Board of Regents for further instructions. The hope was informally expressed that they might be published at least in summary or in a condensed form.
3) That an additional (or third) committee be authorized by the Board of Regents to function under this Council. This new committee will be specifically interested in hospital practices dealing with such matters as personnel, salaries, operation costs, etc., etc. It will be required from time to time to seek data on such items for the information of administrators, hospital boards, and the like.
4) That more time be given the open session of the Council at future meetings of the College, an entire session if feasible.

Your chairman shares the opinion held by the members of the Council that the project of setting up minimum standards for sanatoria is a most important and equally exacting one. There is still more work to be done on this assignment before definitive recommendations can be presented to the College. In fact, it will be next to impossible to set up a completed code of standards unless the Committee on Standardization or some special committee can meet at some central point and cover the various items together for a period of several days.

This report was adopted by the Board of Regents.

Committee on College By-Laws

Dr. Charles M. Hendricks, Chairman of the Committee, reported that the revision of the College By-Laws had been completed and that copies of the revised By-Laws will be sent to the members of the Board of Regents through the mails. The Board of Regents will act on the report of the Committee at their semi-annual meeting to be held in Miami Beach, Florida on Saturday, October 23, 1948. Upon approval of the By-Laws by the Board of Regents, the By-Laws will be published in the official journal of the College, "Diseases of the Chest," and acted upon by the members at the next annual meeting of the College to be held in Atlantic City, June 2-5, 1949.

Council on Research

Dr. Alvan L. Barach, Chairman of the Scientific Section of the Council, reported on several projects which were being studied. These projects were referred to special committees for further study.

Committee on the Management and Treatment of Diseases of the Chest

Dr. Edwin R. Levine, Chairman, presented the report of the committee. It was recommended that the committee be changed to a council and this recommendation was referred to the Committee on College By-Laws. Dr. Levine then introduced Dr. Harold G. Trimble, Chairman of the Committee on Non Surgical Collapse Therapy, who discussed the questionnaire on standardization of treatment being circulated among a selected group of physicians in the United States and other countries. It is the intention of the committee to extend the survey to the College chapters in order to obtain the opinions of a larger group. The information will then be compiled for publication. This report was approved by the Board of Regents.

Committee on Chest Diseases in Penal and Mental Institutions

The report of the activities of this committee was presented by Dr. Otto L. Bettag, Chairman. The report was well received and a vote of thanks was extended to Dr. Bettag for the excellent work of the committee. The report was approved and will be published in a later issue of the journal.

Council on International Affairs

Dr. Chevalier L. Jackson reported for the Council on Pan American Affairs, Dr. Andrew L. Banyai for the Council on European Affairs, and Dr. Harry C. Warren for the Council on Pan Pacific Affairs. The Board of Regents was pleased with the progress being made by each of these councils and a vote of thanks was accorded to each chairman. Complete reports of the activities of the above councils will be published in a later issue of the journal.

Council on Undergraduate Medical Education

Dr. Edward W. Hayes, Chairman of the Council, reported that the first set of galleyproofs for the book entitled "The Fundamentals of Pulmonary Tuberculosis and Its Complications" has been submitted to the authors and the publishers have promised to have the book available for distribution within the next few months.

Dr. Andrew L. Banyai, Chairman of the Editorial Committee for the book on non-tuberculous diseases of the chest submitted the following report:

As authorized by the Board of Regents of the College, our committee has proceeded to organize the book on non-tuberculous diseases of the chest which we hope will be a fitting companion to the book now on the press entitled "The Fundamentals of Pulmonary Tuberculosis and Its Complications." The following well-known authors have agreed to write on the subjects indicated:

Roland V. Christie, M.D., London, England: Emphysema
Louis H. Clerf, M.D., Philadelphia, Pennsylvania: Benign Tumors

George M. Curtis, M.D., Columbus, Ohio: Traumatic Diseases of the Lung and Pleura
Seymour M. Farber, M.D., San Francisco, California: Carcinoma
Louis L. Friedman, M.D., Birmingham, Alabama: Diseases of the Pleura, including Spontaneous Pneumothorax
Alvis E. Greer, M.D., Houston, Texas: Fungus Diseases of the Lung
Charles M. Hendricks, M.D., El Paso, Texas: Bronchiectasis
Chevalier L. Jackson, M.D., Philadelphia, Pennsylvania: Foreign Bodies in the Lung
Minas Joannides, M.D., Chicago, Illinois: Diseases of the Diaphragm
Edwin R. Levine, M.D., Chicago, Illinois: Bronchitis and Bronchiolitis
Edgar Mayer, M.D., New York, N. Y.: Pneumonoconiosis
Herman J. Moersch, M.D., and Associate, Rochester, Minnesota: Infarction and Embolism
Jay Arthur Myers, M.D., Minneapolis, Minnesota: Abscess of the Lung
Richard H. Overholt, M.D., Brookline, Massachusetts: Diseases of the Mediastinum
Richard H. Overholt, M.D., and Associate, Brookline, Massachusetts: Cystic Disease of the Lung
George G. Ornstein, M.D., New York, N. Y.: Pulmonary Function
J. Winthrop Peabody, M.D., Washington, D. C.: Loeffler's Syndrome
Bret Ratner, M.D., New York, N. Y.: Bronchial Asthma in Children
Gumersindo Sayago, M.D., Cordoba, Argentina: Echinococcus Disease of the Lung
J. J. Singer, M.D., Los Angeles, California: Sarcoidosis
Leon Unger, M.D., Chicago, Illinois: Bronchial Asthma in Adults
Raman Viswanathan, M.D., New Delhi, India: Tropical Diseases of the Lung
Italo Volini, M.D., Chicago, Illinois: The Pneumonias
Andrew L. Banyai, M.D., Milwaukee, Wisconsin: Miscellaneous Topics

No effort or time will be spared in making this book as complete and informative as technical limitations permit. Additional plans are being carried out to achieve this goal. We hope to have the manuscripts completed and in the hands of the printer by the end of this year.

Council on Postgraduate Medical Education

Dr. J. Winthrop Peabody, Chairman of the Council, reports on the postgraduate course held in Chicago in September, 1947 and in Philadelphia, March, 1948. Both of these courses were highly successful and the Council has completed plans for three postgraduate courses to be presented in the fall of this year. A postgraduate course in diseases of the chest will be given in San Francisco in connection with the University of California Medical School, September 13-17; a second course in diseases of the chest will be given in Chicago, September 20-25; and a third postgraduate course in diseases of the chest will be presented in New York City, November 8-12. The report of the Council on Postgraduate Medical Education was approved with thanks.

Conference of College Chapter Officials

Dr. Seymour M. Farber, Secretary of the Conference, reported that at their meeting held in Chicago on June 17, the Conference adopted a proposition that the Board of Regents find ways and means of financing speakers to chapter and sectional meetings of the College. In view of

the fact that the national organization is operating at a deficit at the present time, it was decided that it would be inadvisable for the College to attempt to finance the traveling expenses of speakers to chapter and sectional meetings at this time. It was recommended that the College revive its Speakers Bureau and obtain the names of such speakers who would be willing to travel to chapter and sectional meetings at their own expense.

Resolutions

A resolution was introduced by the Board of Governors recommending the clarification of the processing of applications for membership in the College. It was voted to refer this matter to the Committee on College By-Laws.

A resolution was introduced recommending that the Board of Regents hold their semi-annual meeting in Miami Beach on October 23, 1948. The Southern Chapter of the College will meet with the Southern Medical Association in Miami, October 24-25. An invitation has been extended by the Cuban Chapter of the College for the College members attending the meetings in Miami to meet with them in Havana, Cuba on October 26. The resolution was approved.

Communications

A letter was presented from the New Jersey Chapter of the College regarding the schedule of fees for pneumothorax refills advocated by the Veterans Administration, and the Veteran's right to a free choice of physicians. This matter was referred to the Executive Council for further study.

Announcement was made in accord with the College By-Laws that the 15th Annual Meeting of the College will be held in Atlantic City, June 2-5, 1949. The American Medical Association will hold its annual meeting in Atlantic City, June 6-10, 1949.

Announcements

Dr. William E. Ogden, Regent of the College for Canada, commented on the growth of the College membership in Canada and in view of the large expanse of the country, he recommended that members in the various Canadian Provinces meet with College chapters in the United States adjacent to their territories, rather than organize independent College chapters. This proposal was approved by the Board of Regents of the College.

Dr. Donald R. McKay, alternate for New York State, announced the establishment of a Section on Diseases of the Chest in the Medical Society of the State of New York. Dr. McKay expressed the regrets of Dr. Nelson W. Strohm, Regent of the College for New York State, at not being able to be present at the meeting in Chicago.

New Officers

Dr. Robert B. Homan, El Paso, Texas, was elected by the Board of Regents as a member of the Committee on Nominations.

Dr. James H. Stygall, Indianapolis, Indiana, was elected by the Board of Regents to the Executive Council.

Dr. Paul A. Turner, Louisville, Kentucky, was elected as Chairman of the Board of Regents to succeed Dr. Joseph C. Placak, Cleveland, Ohio,

who is now President-Elect of the College.

Dr. William A. Hudson, Detroit, Michigan, was re-elected as Historian of the College.

Section on Diseases of the Chest in A.M.A. Draws Large Audience

The first Session on Diseases of the Chest in the newly established Section on Diseases of the Chest in the Scientific Assembly of the American Medical Association was presented at Navy Pier, Chicago on Thursday, June 24. More than 500 physicians attended the session at which a symposium on streptomycin was presented by leading authorities. The papers were well received and the concensus was that this symposium on streptomycin gave to the medical profession the latest information on the use of the drug in the treatment of various chest conditions.

Following the reading of the first scientific paper, the Nominating Committee, appointed by Dr. Richard H. Overholt, Chairman of the Session, comprised of Dr. Edward W. Hayes, Monrovia, California, Dr. Andrew L. Banyai, Milwaukee, Wisconsin, and Dr. Joseph C. Placak, Cleveland, Ohio, presented the following slate of officers for the Section on Diseases of the Chest:

S. U. Marietta, M.D., Washington, D. C., Chairman
Harry C. Warren, M.D., San Francisco, California, Vice-Chairman
Jay Arthur Myers, M.D., Minneapolis, Minnesota, Secretary
J. Winthrop Peabody, M.D., Washington, D. C., Delegate
Charles M. Hendricks, M.D., El Paso, Texas, Alternate

The above officers were unanimously elected by the physicians present who were registered in the Section on Diseases of the Chest. The House of Delegates and the Council of the American Medical Association are to be congratulated for the establishment of this Section on Diseases of the Chest in the Scientific Assembly.

"Diseases of the Chest" to be Published Monthly

The Editorial Board has been unable to publish the large number of timely papers submitted for publication in "Diseases of the Chest" despite the increase in the number of pages in the journal during the past year. Because of this situation, the Editorial Board recommended to the Board of Regents of the College that the journal be published monthly instead of bi-monthly. The Board of Regents voted favorably on this proposal and commencing with the January, 1949 issue, "Diseases of the Chest" will become a monthly publication.

The increase in the cost of publishing the journal monthly, in addition to the increase in all other College expenses, will result in a deficit of $25,000 for the year 1949. In order to partially meet this deficit, the Board of Regents has voted to increase the annual dues for Fellows of the College from $15.00 to $25.00, and for Associate Fellows and Associate Members from $15.00 to $20.00. This increase in dues applies to members residing in the United States of America and is effective January 1, 1949. The subscription price for "Diseases of the Chest" will be advanced from $5.00 to $8.50 per year to subscribers in the United States, and from $6.00 to $9.50 per year to subscribers in other countries. This increase in subscription becomes effective January 1, 1949.

Announcement

The scientific papers presented at the 14th Annual Meeting of the College held in Chicago, will be published in future issues of "Diseases of the Chest."

Report of the Historian

Mr. President, ladies, and distinguished guests and Fellows of the American College of Chest Physicians:

It is fitting and proper that at this time in our deliberations we pause to pay respect to the memory of our fellow physicians who have completed their earthly duties and have passed to their reward.

The example which they gave to us, their fellow physicians, shall live forever. The seeds which they sowed with such copious hands shall still germinate and bear fruit under the full light of heaven.

Their admonition to us is: "Finish every day and be done with it. You have done what you could. Some blunders and absurdities may have crept in, forget them as soon as you can. Tomorrow is a new day. You shall begin it well and serenely and with too high a spirit to be encumbered with your old nonsense."

"Their moving fingers writ and having written moved on, nor all our pity nor wit shall lure them back to cancel half a line, nor all our tears wash out a word of it."

DECEASED MEMBERS

June 1, 1947 — June 1, 1948

Max Pinner, M.D., Berkeley, California,
Editor of the American Review of Tuberculosis
Pierre Ameiulle, M.D., Paris, France
George Cambers Anglin, M.D., Toronto, Ontario, Canada
Henry C. Drew, M.D., Washington, D. C.
Eugenio Garcia Fernandez, M.D., Hato Rey, Puerto Rico
William Gregory Gunn, M.D., Versailles, Missouri
Frank T. Harper, M.D., Burlington, North Carolina
Harwood LeRoy Hollis, M.D., Lacona, New York
Wilson Pendleton, M.D., Asheville, North Carolina
Harry Patton Reid, M.D., Legion, Texas
Phillip Schonwald, M.D., Seattle, Washington
Bryant R. Simpson, M.D., San Diego, California
Samuel Humes Watson, M.D., Tucson, Arizona
Walter Hobert Watterson, M.D., LaGrange, Illinois
Leon Gilbert Woodford, M.D., Everett, Washington

* * * * *

William Devitt, M.D., Allenwood, Pennsylvania

Our beloved first president, 1935-1937, a kindly man whose heart was filled to overflow with sympathy and tender solicitude for all who were afflicted with infirmities. His was a keen mind filled with visions of things to come. He well foresaw a great future for our College.

Time goes on, you say? Ah, no, alas time stays, we go, leaving behind our ever act, thought, and word to reverberate in the halls of time through all eternity.

Respectfully submitted, William A. Hudson, M.D., F.C.C.P.

Convocation

The Third Annual Convocation was held at the time of the 14th Annual Meeting of the College and 129 candidates were admitted to Fellowship in the class of 1948. The Fellowship Certificates were awarded to the new Fellows by Major General S. U. Marietta, Washington, D. C., President of the College. The Convocation address was delivered by Dr. J. Roscoe Miller, Chicago, Illinois, Dean of Northwestern University Medical School. Dr. Miller was introduced by Dr. Richard H. Overholt, Brookline, Massachusetts, President-Elect of the College.

The following candidates were admitted to Fellowship in the College in the class of 1948, and received their Fellowship Certificates:

Osler A. Abbott, Atlanta, Georgia
Alfred Adler, Mt. Vernon, Missouri
Reuben M. Anderson, Hackensack, New Jersey
Albert H. Andrews, Chicago, Illinois
Joseph P. Atkins, Wynnwood, Pennsylvania
Albert H. Baker, Calgary, Canada
Ralph W. Ballin, State Sanatorium, Maryland
William S. Barclay, Sardis, Canada
Solomon S. Bauch, Newark, New Jersey
Lewis F. Baum, South Orange, New Jersey
Mary C. Block, Santa Ana, California
Johann Bornstein, Chicago, Illinois
Henry J. Brock, Buffalo, New York
Cabot Brown, San Francisco, California
H. H. Brueckner, Canton, Ohio
J. Edmond Bryant, Evanston, Illinois
L. James Buis, Richmond, Virginia
J. J. Burrascano, New York, New York
William S. Burton, Richmond, Virginia
Robert O. Canada, Washington, D. C.
A. A. Carabelli, Trenton, New Jersey
Sumner S. Cohen, St. Louis Park, Minnesota
Charles B. Craft, Bozeman, Montana
Eugene J. DesAutels, Hines, Illinois
J. Edward Dolan, Perry Point, Maryland
Thomas E. Dredge, Minneapolis, Minnesota
Isaac Epstein, Alexandria, Louisiana
David D. Feld, Milwaukee, Wisconsin
John P. Fetherston, Milwaukee, Wisconsin
Y. F. Fujikawa, Mt. Vernon, Missouri
Andre Gelinas, St. Hyacinthe, Canada
William H. Glass, Hartford, Connecticut
Jacob Goldberg, Castle Point, New York
Leon Goldberg, New York, New York
Leon H. Gornel, Los Angeles, California
George H. Hames, Saskatoon, Canada
Irving H. Herman, Saskatoon, Canada
Gerald F. Hogan, Amherst, Massachusetts
Isaac Horowitz, Brooklyn, New York
Hugh L. Houston, Murray, Kentucky
W. Leonard Howard, Northville, Michigan
George R. Howell, Montreal, Canada
Leroy Hyde, Van Nuys, California
Linneus G. Idstrom, Minneapolis, Minnesota
Kenneth C. Johnston, Chicago, Illinois
Archibald R. Judd, Hamburg, Pennsylvania
Gordon F. Kincade, Vancouver, Canada
Karl P. Klassen, Columbus, Ohio
William S. Klein, Chicago, Illinois
Alexander Krasnitz, New Lisbon, New Jersey
Robert C. Laird, Toronto, Canada
Aaron A. Landy, Van Nuys, California

S. A. Levinson, Chicago, Illinois
Anthony D. Lioy, Talihina, Oklahoma
Harold R. Lipscomb, Aspinwall, Pennsylvania
Ellert E. Lundegaard, Orange, California
Harold A. Lyons, Brooklyn, New York
Saul A. Mackler, Chicago, Illinois
Edward H. Mandell, Minneapolis, Minnesota
David Marcus, Cleveland, Ohio
Alexander Marshall, Tranquille, Canada
William K. Massey, Nelson, Canada
Bernard E. McGovern, North Hollywood, California
Clifford C. McLean, Kitchener, Canada
Melvyn McQuitty, Ste. Anne de Bellevue, Canada
Patrick McShane, New Brunswick, New Jersey
Constantine P. Mehas, Pontiac, Michigan
Edwin Mendelsohn, Philadelphia, Pennsylvania
Gerard Michaud, Roberval, Canada
Philip Morgenstern, Black Mountain, North Carolina
Robert B. Morrison, Austin, Texas
Solomon Netzer, San Fernando, California
William E. Nutzman, Kearney, Nebraska
John A. O'Hale, Brecksville, Ohio
Francis H. O'Neill, Pittsburgh, Pennsylvania
Forrest M. Ostrander, Battle Creek, Michigan
Jean P. Paquette, Montreal, Canada
Harry E. Peart, Hamilton, Canada
Samuel Phillips, Memphis, Tennessee
Irving Pine, Asheville, North Carolina
Joseph C. Placak, Jr., Cleveland, Ohio
Virgil A. Plessinger, Cincinnati, Ohio
Sam Poller, Castle Point, New York
Henkel M. Price, Martinsville, Virginia
Solomon M. Rauchwerger, Oteen, North Carolina
Leo G. Rigler, Minneapolis, Minnesota
John J. Quinlan, Kentville, Canada
Alfred Ring, Jamaica, New York
James A. R. Rogers, Patterson, New Jersey
Paul Wolfe Roman, Baltimore, Maryland
George C. Roth, St. Paul, Minnesota
Wyatt E. Roye, Richmond, Virginia
Jack H. Rubin, Outremont, Canada
Charles W. Rudolph, Tucson, Arizona
Robert E. Schell, Swannanoa, North Carolina
Miller H. Schuck, Buffalo, New York
John A. Seaberg, Minneapolis, Minnesota
Reuben I. Shapiro, Detroit, Michigan
David V. Sharp, Minneapolis, Minnesota
J. Vincent Sherwood, Grand Rapids, Michigan
Maurice M. Shoor, Los Angeles, California
Harry Shubin, Philadelphia, Pennsylvania
Alexander C. Sinclair, St. Vital, Canada
Bertram L. Snyder, Phoenix, Arizona
Robert M. Sonneborn, Wheeling, West Virginia
Aaron A. Sprong, Excelsior Springs, Missouri
Herbert S. Stalker, Tranquille, Canada
Reuben E. Stone, Washington, D. C.
J. Earle Stuart, Plainfield, New Jersey
Lloyd K. Swasey, Phoenix, Arizona
Henry C. Sweany, Chicago, Illinois
John T. Szypulski, Mt. Carmel, Pennsylvania
Henry K. Taylor, New York, New York
Joseph F. Tedesco, Castle Point, New York
Efton J. Thomas, Miami Beach, Florida
Samuel Topperman, Otisville, New York
Kenneth A. Tyler, Gooding, Idaho
Robert A. Ullman, Buffalo, New York
Joseph L. Versage, Stockertown, Pennsylvania

Italo F. Volini, Chicago, Illinois
Agnes M. Walker, Hamilton, Canada
Starnes E. Walker, Kansas City, Missouri
Henry W. Walters, Sunmount, New York
Francis J. Weber, Washington, D. C.
Clarence L. Wheaton, Chicago, Illinois
Gertrude H. Wilber, Great Kills, New York
Francis M. Woods, Brookline, Massachusetts
Cuthberg B. Young, Tyler, Texas
Julius Zelman, San Bernardino, California

College Chapter News

CALIFORNIA CHAPTER

Cabot Brown, M.D., F.C.C.P., San Francisco, President of the California Chapter of the College, has announced the following committee appointments for the ensuing year:

Membership Committee

John C. Sharp, M.D., Salinas, Chairman
William A. Cassidy, M.D., Livermore
Rudolph H. Sundberg, M.D., San Diego

Program Committee

Lyman A. Brewer, III, M.D., Los Angeles, Chairman
Gordon A. Diddy, M.D., Ahwahnee
Joseph L. Robinson, M.D., Los Angeles

Nominating Committee

Edward W. Hayes, M.D., Monrovia, California
Forrest J. Bell, M.D., San Francisco
Jacob J. Singer, M.D., Beverly Hills

Postgraduate and Undergraduate Education Committee

Frank S. Dolley, M.D., Los Angeles, Chairman
Seymour M. Farber, M.D., San Francisco
Edward W. Hayes, M.D., Monrovia
William L. Rogers, M.D., San Francisco

NEW ENGLAND STATES CHAPTER

The New England States Chapter of the College held its annual meeting on July 10 at the State Sanatorium, Wallum Lake, Rhode Island. The following program was presented:

"Diagnostic Bronchial Lavage in Tuberculosis,"
Marcio M. Bueno, M.D., F.C.C.P., Fall River, Massachusetts

"Pathological Bronchoscopic and Surgical Aspects of Endobronchial Tuberculosis,"
Norman Wilson, M.D., Boston, Massachusetts

PENNSYLVANIA CHAPTER

The Pennsylvania Chapter of the College will hold its annual business and scientific meeting in Philadelphia on Monday, October 4. Further details will be sent to the members of the chapter regarding reservations. The annual meeting of the Medical Society of the State of Pennsylvania will be held in Philadelphia, October 4-7.

WISCONSIN CHAPTER

The annual meeting of the Wisconsin Chapter of the College will be held in Milwaukee on Sunday, October 3rd. An interesting scientific program is being planned and will be announced in a later issue of the journal.

TUBERCULOSIS SOCIETY OF ROSARIO, ARGENTINA

The new officers of the Tuberculosis Society of Rosario, Argentina are as follows:

Juan Carlos Barberis, M.D., President
Abraham F. Schottlender, M.D., General Secretary
Jorge Geary, M.D., Acting Secretary
Federico Scharping, M.D., Treasurer
Manuel B. Pardo, M.D., Angel Invaldi, M.D., and
Isaac Alberto Hassan, M.D., Directors

Drs. Schottlender, Pardo and Hassan are members of the American College of Chest Physicians.

College News Notes

Hollis E. Johnson, M.D., F.C.C.P., Nashville, Tennessee, has been appointed by the President of the College as Regent for District No. 9 comprising the states of Alabama, Kentucky, Louisiana, Mississippi and Tennessee, to complete the unexpired term of Paul A. Turner, M.D., F.C.C.P., Louisville, Kentucky, who has been elected as Chairman of the Board of Regents of the College.

On February 1, 1948, Miguel Jimenez, M.D., F.C.C.P., Mexico City, Mexico, was appointed the Director of Tuberculosis for the Republic of Mexico.

Bret Ratner, M.D., F.C.C.P., Clinical Professor of Pediatrics, New York University College of Medicine, was the principal speaker at a dinner meeting of the Philadelphia Allergy Society on February 25 in Philadelphia. His address was entitled "Pseudodoxia Allergica" in which Dr. Ratner called attention to the many false opinions and false practices in allergy today.

William A. Hudson, M.D., F.C.C.P., Detroit, Michigan, gave a lecture on "Surgery of Pulmonary Tuberculosis" before the senior class of the University of Arkansas Medical School on April 22 and on the same evening lectured before a group of surgeons of Little Rock (and Pulaska County, Arkansas) on "Diagnostic and Surgical Problems in Thoracic Surgery; Special Attention to Tumors of the Thorax." On April 30, Dr. Hudson gave the Frank Vinsonhaler Memorial Lecture before the students, faculty and guests at the University of Arkansas Medical School. His subject was "A Historical Review Concerning the Development of Thoracic Surgery."

Manuel Albertal, M.D., F.C.C.P., Buenos Aires, Argentina, a member of the Committee on Chemotherapy and Antibiotics of the College, was invited to address the Cuban Chapter of the College at Havana on July 5, the Venezuelan Chapter on July 7, and the Peruvian Chapter on July 9. Dr. Albertal will speak to the members of these College Chapters on the recent developments in the treatment of chest diseases with streptomycin. While in the United States, Dr. Albertal made an extensive tour of the various hospitals where streptomycin is being used in the treatment of chest diseases and he carries with him to the Argentine the latest information on this subject. He participated in the program of the 14th Annual Meeting of the College held in Chicago, June 17-20. Dr. Albertal will return to Buenos Aires on July 10th.

BOOKS AND MEDICAL JOURNALS WANTED FOR CHINA

The Council on Pan Pacific Affairs of the College will appreciate receiving old or current medical books and journals for shipment to medical schools in China. Please send your books and journals to Dr. Harry C. Warren, Chairman, Council on Pan Pacific Affairs, American College of Chest Physicians, 384 Post Street, San Francisco, California.

FALL POSTGRADUATE COURSES

The American College of Chest Physicians announces the presentation of three postgraduate courses in diseases of the chest being sponsored by the Council on Postgraduate Medical Education of the College. The courses will be held in San Francisco, Chicago and New York City. Further information regarding these courses and coupon for application may be found on page x in the front advertising section of this issue of the journal.

AMERICAN COLLEGE OF PHYSICIANS ANNOUNCES POSTGRADUATE COURSE IN CARDIOLOGY

The American College of Physicians has announced a Postgraduate Course in Cardiology to be held at the National Institute of Cardiology of Mexico, Mexico City, D. F., August 2-13, 1948. Registration for the course must be made through the Executive Offices of the American College of Physicians, 4200 Pine Street, Philadelphia 4, Pennsylvania.

Book Review

Exercise During Convalescence, by George T. Stafford, Ed. D., Professor of Physical Education, University of Illinois. A. S. Barnes and Co., New York, 1947.

In recent years there has been an increasing interest in the convalescent phase of disease and a realization that the period during which a patient is returning to maximum physical and mental capability is as much a matter of medical concern as the treatment of the acute phases. During World War II, these studies received added impetus as a result of the acute necessity for restoring patients to peak condition as rapidly as possible. The war time findings have since been found applicable to civilian conditions. However, in many tuberculosis institutions attention is still focussed completely on cavity closure and sputum conversion without serious regard for the individual's rehabilitation and with no concern for his ability to resume reasonably normal existence on leaving the institution.

This brief work serves a valuable purpose in pointing out the need for rehabilitation as an integral part of any therapeutic program. Although the author concerns himself primarily with physical exercise, he states that a complete program "employs physical therapy, occupational therapy, therapeutic exercises, and recreational activities, sheltered and curative workshops, and other therapeutic and social services to restore the patient to his highest level of total fitness." It is necessary that the physician, the social service department, physical and occupational therapists, vocational counsellors, and others work together within the framework of an integrated program which is individually adapted to each patient's needs. Only when one observes such coordinated program in operation does one realize that anything less is inadequate treatment. Such rehabilitation must "begin when the diagnosis is made, should continue throughout his sanatorium and into the post sanatorium readjustment period as long as necessary." Concentration on the pulmonary disease while ignoring the individual's total preparedness for post-hospital existence is a dereliction of our duty to the patient, his family, and the community at large.

The bulk of Dr. Stafford's book is devoted to a description of physical exercises suitable for various types of disability. In one short chapter devoted to tuberculosis, he describes various exercises that may be used in a graduated fashion by the tuberculous convalescent. The list is necessarily incomplete, and does not include some of the familiar ones employed in reconditioning such special cases as the post thoracoplasty patient. However, these will quickly suggest themselves to any one familiar with the fundamentals of physical medicine.

In summary, this book is valuable because it emphasizes the need for beginning rehabilitation early, and provides a basis for a physical therapy program.

Obituaries

WILLIAM DEVITT

1874 - 1948

Dr. William Devitt was born in Philadelphia in 1874, and died at his home at Devitt's Camp, Allenwood, Pennsylvania, on May 20, 1948. Forced to leave school at the age of thirteen, he became a breadwinner in the mills of Manayunk, one of the industrial sections of Philadelphia. He attended Temple University at night and worked his way through Bucknell Academy. He next attended the Medico-Chirurgical College, now the graduate medical school of the University of Pennsylvania and graduated in 1902. He began practicing in Manayunk.

In April, 1912, he established the institution which has since been known as Devitt's Camp, a private tuberculosis sanatorium, at Allen-

wood, Pennsylvania, and gradually built it up into a modern sanatorium. In contrast to the early days when the accommodations were almost primitive, the valuation of the institution is now close to $400,000.00 and accommodates 105 patients with a resident staff of physicians and nurses. It serves referring physicians and their patients for a 200 mile radius.

Dr. Devitt was a Fellow and first President of the American College of Chest Physicians, having been elected at its founding in 1935. In 1940 he received a Certificate of Merit from The Board of Regents of the College commending him for his work as first President.

He was a member of the Lycoming County Medical Society, Pennsylvania Medical Society, Fellow of the American Medical Association and American College of Physicians, Member of the National Tuberculosis Association, International Tuberculosis Association, Medical Club of Philadelphia, Laennec Society of Philadelphia, American Trudeau Society, and certified by the American Board of Internal Medicine. He was a member and Past President of the Pennsylvania Tuberculosis Society. In 1928, Dr. Devitt received the honorary degree of Doctor of Science at Bucknell University. He was a 33 degree Mason.

Dr. Devitt was widely known in Pennsylvania and throughout the country for his pioneer efforts in the fight against tuberculosis. He was always a source of inspiration to his patients, to his colleagues and to his staff members. His kindliness and courage in the face of any difficulty endeared him to all who knew him. The fruits of his work will continue to benefit many through the years to come. As one who was a pupil and later an associate of Dr. Devitt for almost 20 years, I can speak wholeheartedly and sincerely for his hundreds of patients, friends and colleagues.

John S. Packard, M.D., F.C.C.P., Allenwood, Pennsylvania.

WILSON PENDLETON

1886 - 1948

Dr. Wilson Pendleton was born on February 22, 1886 in Portsmouth, Virginia. He received his degree of Doctor of Medicine from the University of Virginia in the class of 1908. During World War I, Dr. Pendleton served as a Captain in the Medical Corps. Dr. Pendleton practiced medicine in Asheville, North Carolina for many years, specializing in diseases of the chest, and latterly in allergy. He was a Fellow and Charter Member of the American College of Chest Physicians.

Dr. Pendleton died suddenly on March 28 of a cerebral hemorrhage.

Merle D. Bonner, M.D., Governor for North Carolina.

RESEARCH FELLOWSHIPS
THE AMERICAN COLLEGE OF PHYSICIANS

The American College of Physicians announces that a limited number of Fellowships in Medicine will be available from July 1, 1949 - June 30, 1950. These Fellowships are designed to provide an opportunity for research training either in the basic medical sciences or in the application of these sciences to clinical investigation. They are for the benefit of physicians who are in the early stages of their preparation for a teaching and investigative career in Internal Medicine. Assurance must be provided that the applicant will be acceptable in the laboratory or clinic of his choice and that he will be provided with the facilities necessary for the proper pursuit of his work.

The stipend will be from $2,200 to $3,200.

Application forms will be supplied on request to The American College of Physicians, 4200 Pine Street, Philadelphia 4, Pa., and must be submitted in duplicate not later than November 1, 1948. Announcement of the awards will be made as promptly as is possible.

DOCTOR DUTCHESS SELECTED TO HEAD NEW A.P.M.A. BOARD

Appointment of Dr. Charles E. Dutchess, medical director of Schenley Laboratories, Inc., as chairman of the newly created board of the American Pharmaceutical Manufacturers Association's medical section has been announced by Dr. Theodore Klumpp, association president.

Dr. Dutchess is a member of the Association for the study of Internal Secretions; the American Society of Tropical Medicine, the Association of Military Surgeons, the Association of Medical Directors, the Pharmaceutical Advertising Club of New York, and is chairman of the public relations committee of the American Association of Industrial Physicians and Surgeons. He is a Fellow of the American Medical Association and the New York Academy of Medicine.

Serving on the board with him are: Dr. J. B. Rice, Winthrop-Stearns, Inc.; Dr. Stanton M. Hardy, Lederle Laboratories; Dr. D. K. Kitchen, Bristol Laboratories; Dr. Paul C. Barton, Brewer and Co., Inc.; Dr. R. L. Conklin, Ames Company, Inc.; Dr. George Hazel, Abbott Laboratories; Dr. Irwin C. Winter, G. D. Searle and Co.; Dr. Paul Spickard, Rexall Drug Co., and Dr. John M. Shaul, Maltbie Chemical Company.

SPECIAL ANNOUNCEMENTS

SEMI-ANNUAL MEETING, BOARD OF REGENTS

The semi-annual meeting of the Board of Regents of the College will be held in Miami Beach, Florida, on Saturday, October 23. A meeting of the Regents and Governors of the College in the Southern States will be held on Sunday morning, October 24.

SOUTHERN CHAPTER MEETING

The Sixth Annual Meeting of the Southern Chapter of the College will be held in Miami, Florida, on October 24 and 25, in connection with the annual meeting of the Southern Medical Association.

An interesting scientific program is being prepared for presentation and luncheon and dinner meetings are also being planned for the meeting. The program for the meeting will be published in the next issue of "Diseases of the Chest."

CUBAN CHAPTER MEETING

The Cuban Chapter of the College has invited College members attending the meeting in Miami to attend a special meeting of the Cuban Chapter in Havana on October 26. College members from the United States will appear on the scientific program, as well as several members of the Cuban Chapter.

College members who plan to attend the meeting in Miami are urged to make arrangements for a few day's visit to Havana in order to attend the meeting planned by the Cuban Chapter, and to enjoy sightseeing in the beautiful capital city of Cuba. Arrangements for the trip may be made through the Executive Offices of the College (see coupon below).

ULAST TO MEET IN MEXICO CITY IN 1949

The Union of Latin American Societies on Tuberculosis (ULAST) will hold its bi-annual Congress in Mexico City, January 24-29, 1949. The American College of Chest Physicians has been invited to participate in this Congress and all College members are cordially invited to attend. The Governors and Regents of the College in all of the Latin American and North American countries who attend the Congress will gather at a breakfast meeting on Tuesday, January 25. Later in the week a scientific program will be presented by College members and various other interesting activities are being planned.

A complete program of the Congress will be published in a later issue of the journal. College members who are interested in attending the Congress may obtain more detailed information by returning the coupon at the bottom of this page to the Executive Offices of the College.

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AMERICAN COLLEGE OF CHEST PHYSICIANS
500 North Dearborn Street, Chicago 10, Illinois.

Gentlemen: I am planning to attend the meeting(s) checked below. Please send me all available information.

..... Meeting, Cuban Chapter, American College of Chest Physicians, Havana, Cuba, October 26, 1948.

..... Meeting, Union of Latin American Societies on Tuberculosis, Mexico City, January 24-29, 1949.

Name .....

Address .....

City ..... State .....

Medical Service Bureau

POSITIONS AVAILABLE

Junior physician wanted for sanatorium in New England. Single or married without children, salary and maintenance, temporary license obtainable. For further information please address Box 177A, American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois.

Medical resident wanted; full maintenance; salary $300.00 per month; 157 bed tuberculosis hospital. For further information please address Box 178A, American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois.

Dietician: Full time, capable and willing to take over entire dietary department. Salary $250.00 per month. Full maintenance. For further information please address Box 179A, American College of Chest Physicians, 500 N. Dearborn St., Chicago 10, Ill.

Physician wanted, experienced in general medicine, sanatorium in Alaska. Complete maintenance, one month's vacation, transportation paid, $500-$700 per month, depending upon training and experience. For further information please address Box 180A, American College of Chest Physicians, 500 N. Dearborn St., Chicago 10, Illinois.

Wanted: Assistant physician for tuberculosis sanatorium. Salary open. Address Medical Superintendent, Stillwater Sanatorium, Dayton 5, Ohio.

Position available: Resident physician for tuberculosis sanatorium, experience available in all forms of collapse therapy. Credit obtainable toward one year's requirement for Board of Internal Medicine. Located in resort area country, beautiful surroundings. Exceptionally complete maintenance for self and family. Within forty miles of two medical universities. Salary dependent on experience. Must have Michigan license. For further information please address Box 181A, American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois.

Position open for a chief resident physician and a junior resident physician in an outstanding tuberculosis institution with over 200 bed capacity. Salary, including maintenance, commensurate with experience and qualifications. For further information please address Box 182A, American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois.

Resident physician wanted for 500-bed sanatorium. Should have at least one year's experience in chest diseases. Complete chest service in teaching hospital of medical school. Salary depending upon experience, including full family maintenance. Waverly Hills Sanatorium, Waverly Hills, Kentucky.

Junior staff physician wanted. Yearly increase in salary, liberal sick time, paid vacation, optional insurance and retirement plan. Must qualify for registration in Michigan. 840 bed capacity, complete service plus research. For further information address Box 183A, American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois.

Assistant superintendent wanted in tuberculosis hospital in south-east Texas. Excellent opportunity. For further information please address Box 184A, American College of Chest Physicians, 500 N. Dearborn St., Chicago 10, Illinois.

Physician in Charge 60-bed institution, capable of assuming responsibilities of an active medical and surgical service. Salary depending on experience and ability, including maintenance. For further information please address Box 185A, American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois.

Pathologist wanted, interested in the development of a research program. Duties include routine and special laboratory work relating to diseases of the chest. Salary commensurate with ability, includes maintenance. For further information please address Box 186A, American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois.

Resident physician wanted. Good salary and maintenance. Prefer young man with family. For further information please address Box 187A, American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois.